Volume 43 of the Nebraska Symposium on Motivation

Perspectives on Anxiety, Panic, and Fear

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Perspectives on Anxiety, Panic, and Fear is Volume 43 in the series
CURRENT THEORY AND RESEARCH
IN MOTIVATION

The passage quoted on pages 73–77 is from
The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system, by J. A. Gray, 1982, Oxford: Oxford University Press. Reprinted by permission of Oxford University Press.

Manufactured in the United States of America
International Standard Book Number
0-8032-2382-X (Clothbound)

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"The Library of Congress has cataloged
this serial publication as follows:"
Nebraska Symposium on Motivation.
Nebraska Symposium on Motivation.
[Papers] v. [1]–1953–
Lincoln, University of Nebraska Press.
v. illus., diagrs. 22cm. annual.
Vol. 1 issued by the symposium under
its earlier name: Current Theory and
Research in Motivation.
Symposia sponsored by the Dept. of
Psychology of the University of Nebraska.
1. Motivation (Psychology)
BF683.N4 159.4082 53-11655
Library of Congress

Conditioning and Ethological Models of Anxiety Disorders: Stress-in-Dynamic-Context Anxiety Models

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Early in this century two starkly contrasting views regarding the origins of fears, phobias, and anxiety states were put forth, one by Freud (1926/1936) and the other by Pavlov (1927) and Watson (Watson & Rayner, 1920). These two views still represent two of the major different ways psychologists think about the origins of fears and anxiety disorders even today—although now there are also biological as well as cognitive approaches. According to Freud and other theorists of the psychoanalytic tradition, anxiety is viewed as a reaction to, and a signal of, unconscious memories of real or imagined dangers that are often associated with infantile wishes (1926/1936). By contrast, according to Pavlov and Watson, fears and anxiety disorders were seen as arising out of simple classical conditioning of fear when neutral objects

The authors would like to thank the following individuals for their comments on an earlier version of this chapter: Caroline Blanchard, Dianne Chambless, Robert Hendersen, and Steve Maier. We are particularly indebted to Robert Hendersen for suggesting the terms "Stress-in-Total-Isolation Anxiety models" and "Stress-in-Dynamic-Context Anxiety models."

or situations have been paired with trauma. Later theorists in this tradition such as Mowrer (1947, 1960) and Eysenck and Rachman (1965) elaborated on this view and developed an avoidance model of these disorders, in which avoidance responses were seen as motivated by classically conditioned fear, and reinforced by fear or anxiety reduction. As the distinctions between the different anxiety disorders became better understood, the avoidance model was seen as especially compelling for specific and social phobias, agoraphobia, and obsessive-compulsive disorder because of the prominent and persistent avoidance behaviors that characterize these disorders.

One apparent attraction of the psychoanalytic view is that it appears to capture, at least in a post hoc fashion, the richness and diversity of factors that go into the origins and maintenance of people's fears and anxieties. Indeed, this rich interpretive framework that it provides has been part of the continuing source of interest in this theory for many clinicians. But an obvious weakness of this view is that it has not put forth many hypotheses or propositions that are scientifically testable through empirical research. The strengths and weaknesses of the behavioral approach of Pavlov, Watson, and others are in some sense the opposite. Because this approach stems from an empirical tradition, many hypotheses and propositions of the behavioral approach have been tested. Unfortunately, some of this research has revealed what appear to be major weaknesses of the original traditional conditioning approaches (e.g., Rachman, 1978, 1990). These weaknesses can be aptly summarized by stating that the early behavioral approaches do not seem adequately to account for the richness and diversity of factors that go into the development and maintenance of people's fears and anxieties.

In this chapter we argue that the central shortcoming of traditional behavioral models has been their failure to consider the dynamic context in which stressors occur in a person's life. By ignoring the powerful effect of dynamic contextual factors on the impact of those stressors, behavioral models have appeared to be much more simplistic than they need to be and to have far less explanatory power than we know they can have. We refer to the more traditional models as Stress-in-Total-Isolation Anxiety models (SITIA models). We will present an alternative model in which we show that stress must always be considered in a dynamic context rather than in isola-

tion in determining the outcome of exposure to stress. The relevant dynamic contextual factors that we will consider include constitutional factors such as temperament, past experiential history (including general factors such as a history of exposure to uncontrollable life events as well as specific factors such as prior traumatic conditioning events), current contextual factors at the time of a stressor (such as whether there are already reliable predictors of the stressor, or the nature of the conditioned stimulus relative to the nature of the unconditioned stimulus, or whether the stressor can be controlled), and future modification of the impact of the stressor (through processes such as forgetting and other memory modifications, as well as through later experiences with other stressors). We refer to these models as the Stress-in-Dynamic-Context Anxiety models (SIDCA models).

The authors of DSM-III-R and DSM-IV (American Psychiatric Association, 1987, 1994) have identified six different anxiety disorders, which share some overlapping features but also have distinctive characteristics. They are specific phobias, social phobias, panic disorder with or without agoraphobia, generalized anxiety disorder, posttraumatic stress disorder, and obsessive-compulsive disorder. In presenting SIDCA models we will review research and thinking from the contemporary conditioning and ethological literatures—both animal and human—that are relevant to understanding important features of each of these disorders. We are particularly attuned to recent developments in conditioning research and theory that greatly expand the scope and explanatory power of conditioning models because they allow the development of SIDCA models. Although none of the research to be presented provides as yet a full-fledged model of the etiology, symptomatology, therapy, and prevention of these disorders as advocated by theorists such as Seligman (1974) and McKinney (1974), we present many interesting and compelling mini-models that can be useful in illuminating certain aspects of many disorders for which full-fledged animal models are not yet or may never be available. Mini-models may be defined as behavioral or cognitive phenomena studied in animals or humans (where the behaviors are experimentally manipulated through either behavioral or physiological manipulations or are carefully and systematically observed, as done by ethologists) that illuminate certain features of the disorder, such as its etiology, *or* its maintenance,

or its symptomatology, *or* its prevention, *or* its treatment (Marks, 1977; Mineka, 1985a). Thus mini-models may help us to understand only one or a few prominent or cardinal features of a disorder; however, it is understood that in reality such factors would act in conjunction or interaction with other factors in producing the full-fledged human disorder. Such interactions form the core of SIDCA models, and although the exact nature of the interactions of causal factors that are likely to be involved in the etiology of these disorders are not always understood at the present time, enough is known to present hypotheses for future research.

When discussing animal models it should be acknowledged that the degree of cross-species similarity on these criteria may depend on the level of analysis one adopts. Substantial differences between species should be expected when focusing on either observable behaviors (e.g., rats commonly defecate when confronted by threat but people rarely do) or the physical properties of the stimuli that are involved in the etiology of the syndromes under investigation (e.g., people sometimes develop anxiety after reading about threat, but we doubt that the written word has ever contributed to the development of anxiety in animals). At the same time, one could reasonably argue that these symptoms that differ at a surface level are produced by underlying processes that are similar across the species. Thus it is reasonable to expect that a conditioned fear response in animals that follows direct traumatic experiences with an aversive stimulus could be argued to be mediated by the same mechanism that mediates the development of anxiety in people after reading about threat. In fact, Wagner's (1979, 1981) Standard-Operating-Procedures theory of conditioning suggests a plausible candidate for such a mechanism—the joint rehearsal in short-term or working memory of the representations of stimuli that had previously been affectively neutral with the representation of a highly aversive stimulus.

Clearly, drawing inferences regarding hypothesized constructs or mechanisms that are not directly observable is more difficult and challenging than simple stimulus-response analyses that focus only on observable variables. By definition, the theorist interested in a latent construct implicitly acknowledges imperfection in his or her measures by drawing a distinction between the latent constructs he or she is interested in and his or her observed measures. Inferences about latent constructs and mechanisms can be on firm footing,

however, by "triangulating" on them—that is, by employing multiple measures (either across studies or within a single study) that are heterogeneous with respect to their sources of bias/error (Campbell & Fiske, 1959; Cook & Campbell, 1979). Thus in our presentation of mini-models of the anxiety disorders, we emphasize underlying constructs and mechanisms. As more information about other differences between the species involved becomes known, it is possible that these mini-models may even come to be regarded as similar to the human phenomenon on all the criteria suggested by Seligman (1974) and McKinney (1974), which include similarities of symptomatology, etiology, therapy, and prevention.

Specific Phobias

Specific phobias involve intense or irrational fears of various objects or situations, which usually lead to a good deal of avoidance behavior. The traditional view of Pavlov and Watson was that phobias are simply intense, classically conditioned fears that result when a neutral object is paired with a traumatic event. By the 1960s, when it became known that classically conditioned fear in the laboratory does not show the high resistance to extinction seen with phobias, theorists such as Eysenck and Rachman (1965) proposed an avoidance learning model of phobia acquisition and maintenance. According to the then prevailing two-process theory of avoidance learning, classically conditioned fear motivates the learning of avoidance responses, which are reinforced or strengthened by a reduction in fear. The apparent superiority of this model arose from the fact that avoidance responses conditioned in the laboratory are notoriously resistant to extinction, as are phobias. As we have argued elsewhere (Mineka, 1985a; Mineka & Zinbarg, 1991; see also Seligman, 1971), however, there are several problems with the avoidance model of phobias and today most theorists favor a modified version of the simpler classical conditioning model (see "Selective Associations," below).

For the past 20 to 25 years, conditioning models of specific phobias have been severely criticized for a variety of reasons (and similar criticisms would apply to the avoidance models). The major problems with the traditional behavioral approaches stem from their having been de-

Table 1

A Summary of the Shortcomings of Stress-in-Total-Isolation Anxiety (SITIA) Models and How They Are Addressed by Stress-in-Dynamic-Context Anxiety (SIDCA) Models

Shortcomings of SITIA Models	SIDCA Model Responses
1. Many cases of phobias and other anxiety disorders have no known traumatic conditioning history.	1. Fear and anxiety can also be acquired through observational conditioning.
2. Many people who do have a traumatic conditioning history do not develop phobias or other anxiety disorders.	2. Temperament and a multitude of experiential variables occurring before, during, or following a traumatic or an observational conditioning experience affect the amount of fear and anxiety that is experienced at the time and that is acquired and maintained into the future.
3. Fears and phobias do not occur to a random arbitrary group of objects or situations.	3. Primates and humans seem to have a biologically based preparedness to more easily associate aversive events with certain objects or situations that were dangerous to our early ancestors.
4. Phobias and other anxiety disorders often appear to be resistant to extinction and may even overgeneralize with the passage of time.	4. (a) Responses conditioned to fear-relevant objects are more resistant to extinction than responses conditioned to fear-irrelevant objects. (b) Conditioned fears that have been extinguished can be reinstated through exposure to noncontingent traumatic events. (c) Simple mental rehearsal of a US can result in increased CR strength. (d) Generalization gradients around a CS+ flatten over time whereas a CS- often loses its fear inhibitory properties over time. (e) Safety signals and avoidance responses may protect a conditioned fear response from extinction.
5. The cognitive processes associated with some anxiety disorders are more complex than traditional models could explain, e.g., the emo-	5. Compound and contextual cues often affect the amount of anxiety and fear that is acquired or displayed; some stimuli may serve as "occasion

Table 1 Continued

Shortcomings of SITIA Models	SIDCA Model Responses
tional reacton to an anxiety/fear trigger is often dependent on contextual factors, and not all cues present during traumatic events become effective CS+s.	setters" that can turn on or turn off conditioned fear even though the occasion setter does not itself elicit or inhibit conditioned fear.
6. Animal models cannot model symptoms that are dependent on self-report (i.e., worry, obsessions, flashbacks, intrusive thoughts, or nightmares).	6. Although some of the surface-level symptoms of anxiety disorders cannot be modeled in animals, it may be possible to model their underlying mechanisms. For example, interpreted within the framework of Wagner's SOP (1981) model of conditioning, conditioned fear responses can model some of the reexperiencing symptoms of PTSD and important aspects of other reexperiencing symptoms.

veloped in the tradition of SITIA models. This was, in part, because Pavlovian methodology dictated that naive animals be placed in soundproof rooms where they were isolated from the experimenter and from noises, smells, or lights that were extraneous to the conditioning process. Today, however, advances in knowledge about the theoretical and empirical foundations of conditioning demand that conditioning models of fear be viewed as a subset of SIDCA models.

The majority of the criticisms of conditioning models from the SITIA tradition can be placed in one of four categories. The first problem comes from clinical observations that in many cases people with these disorders have no known traumatic conditioning history. If so many individuals with fears and phobias have no known traumatic conditioning history, how can we account for the origins of their fears and phobias? The second problem with SITIA conditioning models of phobias is that they have difficulty accounting for why so many individuals who do undergo traumatic experiences do *not* develop phobias. In other words, why do more people with traumatic conditioning histories not have fears and phobias (Rachman, 1990)? The third problem stems from the failure of these models to account for why fears and phobias do not occur to a random arbitrary group of objects or situations associated with trauma, as clearly

explicated by Seligman (1971) in his well-known paper on preparedness and phobias. Finally, a fourth problem with SITIA conditioning models is their failure adequately to account for why some fears are so persistent and why some even increase and overgeneralize with the passage of time. That is, given the laws of extinction of conditioned responses, why are fears and phobias often so resistant to extinction and why do they often generalize to new objects and situations with the passage of time? We now review contemporary conditioning research in the tradition of SIDCA models that resolves each of these problems with SITIA conditioning models. (See Table 1, points 1–4, for a summary of these criticisms.)

VICARIOUS CONDITIONING OF FEARS AND PHOBIAS

How can SIDCA conditioning models account for the origins of fears and phobias in individuals with no known traumatic conditioning history? For some years it has been speculated that vicarious or observational conditioning experiences may play an important role in the origins of some fears and phobias. Numerous studies have shown that human subjects do acquire conditioned responses simply through observing another human model ostensibly receiving shock in the presence of some neutral stimulus (see Green & Osborne, 1985, for a review). These studies, however, have all involved mild conditioning of autonomic responses in single-session laboratory experiments, with no tests for maintenance of the conditioning days or weeks later. Thus these studies do not answer the question of whether strong and persistent fears such as occur in full-blown phobias can also be conditioned observationally. One line of evidence suggesting that they can comes from studies that have asked phobic subjects detailed questions about how their phobias came about. Although retrospective recall is certainly not an infallible measure, the results of several studies provide some support for this hypothesis. For example, Öst and Hugdahl (1981) found that about 17% of specific phobics (40% of animal phobics) recalled a vicarious conditioning incident as having been involved in the origins of their specific phobia. In addition, Merckelbach and associates (1989) found that 53% of their phobics recalled a combination of direct and

vicarious conditioning processes (rather than either alone) as having contributed to the onset of their phobia. Moreover, there is now good experimental evidence stemming from a primate model of fear conditioning that strong and persistent phobiclike fears can be learned through observation alone.

Mineka and Cook and colleagues conducted a series of experiments which demonstrated rapid, strong, and persistent observational conditioning of snake fear in rhesus monkeys (Cook & Mineka, 1987, 1989, 1990; Cook, Mineka, Wolkenstein, & Laitsch, 1985; Mineka & Cook, 1986, 1993; Mineka, Davidson, Cook, & Keir, 1984b). In most of these experiments laboratory-reared young adult monkeys watched unrelated wild-reared monkeys reacting fearfully in the presence of live and toy snakes and nonfearfully in the presence of neutral objects. The lab-reared monkeys did not show a fear of snakes during an initial pretest, and the majority of them rapidly acquired an intense fear of snakes as a result of the observational conditioning sessions (Cook et al., 1985; Mineka et al., 1984b). Indeed, the observers showed levels of fear on the posttest that were nearly as strong as those of the wild-reared monkeys, who had presumably acquired their fear in the wild many years earlier (Mineka, Keir, & Price, 1980). Furthermore, this acquired fear did not show significant signs of diminution over a three-month interval. One additional experiment showed that lab-reared monkeys could acquire the fear simply through watching a color videotape of a model monkey behaving fearfully in the presence of snakes (Cook & Mineka, 1990, Experiment 1). The latter finding is important because it suggests that humans may also be susceptible to acquiring fears vicariously through movies and television rather than simply through observing live models in vivo.

Thus conditioning models from the SITIA tradition paid insufficient attention to the important role that observational or vicarious conditioning can play in the origins of fears and phobias (see Table 1, point 1). In other words, the context of fear conditioning need not include direct experience with an unconditioned traumatic stimulus. Nevertheless, it is also interesting that the combined results of several of these experiments support the hypothesis that the mechanisms involved in observational conditioning may be very similar to those involved in direct traumatic conditioning (see Mineka & Cook, 1993, for a detailed discussion), rather than involving a separate

"pathway" to fear acquisition as has been suggested by Rachman (1978, 1990).

SOURCES OF INDIVIDUAL DIFFERENCES IN THE ACQUISITION OF FEARS AND PHOBIAS

The second problem with SITIA conditioning models is that they fail to account for why not all individuals who undergo traumatic experiences acquire phobias. For example, both Di Nardo, Guzy, and Bak (1988) and Ollendick and King (1991) have noted that many nonphobics have had traumatic experiences in the presence of some potentially phobic objects and yet not acquired a fear or phobia. In addition, Rachman (1978, 1990) argues that conditioning theory cannot account for why so few people acquired lasting fears or phobias during the air raids and bombings of World War II. Although such findings may seem puzzling from the perspective of SITIA conditioning models, they are predicted by SIDCA models (see Table 1, point 2).

Part of the explanation for why only some people who have had traumatic experiences acquire fears and phobias undoubtedly involves genetic and temperamental factors. Theorists ranging from Pavlov to Eysenck and Kagan have noted that personality and temperamental variables affect the dynamics of conditioning and the origins of fear (see Mineka & Zinbarg, 1991, for a review; see also "Social Phobia" section of this chapter). Temperamental variables are incorporated by SIDCA models as part of the dynamic context in which a traumatic or observational conditioning experience occurs but only rarely by SITIA models.

In addition, SIDCA models explicitly acknowledge that much of the variance in the outcome of a traumatic conditioning experience is accounted for by experiential variables that also constitute an important part of the context. Models from the SIDCA tradition note that it is well-known that a multitude of experiential variables occur before, during, or following a traumatic event or an observational fear conditioning experience that can act singly or in combination to affect the amount of fear that is experienced, acquired, or maintained over time (Mineka, 1985a, b; Mineka & Zinbarg, 1991). Thus SIDCA models underscore that to understand why only some people develop fears or phobias, it is important to understand a wide range

of experiential variables on which individuals undergoing the same trauma may differ—one very important aspect of the dynamic context in which conditioning occurs. That this point was not acknowledged by SITIA models may not be too surprising given that traditional Pavlovian methodologies dictated that naive animals be studied in isolated conditioning chambers. Although this methodology was initially developed for good reasons (e.g., to study the fundamentals of conditioning without extraneous potentially confounding variables such as prior experiences on which animals might differ), there were profound dangers hidden in extrapolating directly from the results of such studies to how conditioning experiences might affect humans who were neither "naive" nor isolated from the world during conditioning. SIDCA models explicitly acknowledge such complexities.

One important variable affecting fear conditioning is the amount of prior experience the organism has had with the to-be-conditioned stimulus. For example, the well-known phenomenon of latent inhibition demonstrates that prior exposure to a conditioned stimulus (CS) (before the CS and unconditioned stimulus [US] are ever paired together) reduces the amount of subsequent conditioning to the CS (e.g., Lubow, 1973; Mackintosh, 1983). Using their observational conditioning paradigm, Mineka and Cook (1986) explored whether one would see even stronger attenuation of fear conditioning if the prior exposure to a CS had occurred in the context of watching a nonfearful model monkey behaving nonfearfully with snakes (as opposed to the latent inhibition procedure of simply exposing the observer monkey to the snake without a nonfearful model). Their results showed that prior exposure to a nonfearful monkey behaving nonfearfully with snakes prevented the acquisition of fear on subsequent exposure to a fearful model. Such results may help to account for why correlations between parents' fears and children's fears are not as high as one might expect given our results showing how robust the observational conditioning phenomenon seems to be (Emmelkamp, 1982; Marks, 1987). Extensive preexposure to a nonfearful parent or peer behaving nonfearfully to the phobic object or situation of the other parent may immunize a child against the effects of later seeing the fearful or phobic parent behaving fearfully with that object. Or, put in the context of SIDCA models, people will differ in whether they acquire fear of some ob-

ject during an observational conditioning experience at least partly as a function of their different prior experiences with that object.

A second experiential variable that is of importance in determining individuals' reactions to frightening and traumatic situations is their history of control over important aspects of their environment. Developmental psychologists have long argued that an infant's experience with control over important aspects of its environment promotes secure attachment relationships, exploration of novel events, less fearful reactions to strange or arousing stimuli, and the ability to cope with transient uncontrollable stressful experiences (e.g., Lewis & Goldberg, 1969; Watson, 1979; White, 1959). Evidence supporting this proposition in human infants comes largely from correlational studies. One experiment with infant rhesus monkeys that manipulated control in an experimental fashion over a one-year period, however, supports the importance of early experience with controlling important outcomes in affecting the ability to cope with transient stressful and frightening situations. This study (Mineka, Gunnar, & Champoux, 1986) explored this question by rearing baby monkeys in controllable (Masters) versus uncontrollable (Yoked) environments for the first 10 to 12 months of life. Then the effects of these experiences with control on their reaction to several frightening and novel situations were studied. Their results indicated that Master monkeys who had early experience with control and mastery over appetitive events showed reduced levels of fear, as well as higher levels of exploration in novel situations, relative to Yoked monkeys reared without control. Taken together with the results from the correlational studies with human subjects, these results suggest that individuals with a prior history of control over important events in their environment may show reduced susceptibility to the development of fear and anxiety disorders.

As suggested earlier, experiential variables that occur *during* a conditioning experience, as well as before it, are also an important part of the dynamic context that determines the level of fear that is experienced or conditioned. Historically, Pavlovian or classical conditioning of fear was studied only in paradigms in which the organism had no control over the presentation of the neutral conditioned stimulus or the noxious unconditioned stimulus. The organism was seen as a passive recipient of an essentially involuntary conditioning process. Yet in real life, in many of the everyday events in which

Pavlovian conditioning occurs, organisms do have some control over the US, such as when it will end. And indeed, for some years it has been known that much lower levels of fear are conditioned to neutral stimuli paired with escapable or controllable as opposed to inescapable or uncontrollable shock (e.g., Desiderato & Newman, 1971; Mineka, Cook, & Miller, 1984a; Mowrer & Viek, 1948). In fact, the results of several experiments conducted by Mineka et al. (1984a) indicated that levels of acquired fear in the rats that received uncontrollable shocks were approximately twice as high as levels of acquired fear in rats that received controllable shocks.

There are additional complexities in the dynamics of the conditioning process that overcome some of the problems associated with traditional SITIA conditioning models (see Table 1, point 5). For example, a great deal of recent work has been done on a class of stimuli called "occasion setters" which in and of themselves do not evoke conditioned responding but that may influence behavior in the presence of a CS (Holland, 1983; Rescorla, 1988). That is, the presence or absence of the occasion-setting stimulus can turn on or turn off conditioned fear to a CS even though the occasion setter itself does not evoke fear. In addition, we now know that conditioning does not occur to all stimuli that are present during a traumatic event. Rather, it is the CSs that provide the most reliable and nonredundant information about the occurrence of a US to which conditioning will occur (e.g., Mackintosh, 1983; Rescorla, 1988; Rescorla & Wagner, 1972). Moreover, in a situation where there are multiple potential CSs, the dynamics of which CS has the most strength also vary over time as the relative predictive value of all stimuli present is gradually learned by the organism (Rescorla & Wagner, 1972).

Given these basic facts from contemporary conditioning research, SIDCA models do not lead to the problem of overprediction of fear that Rachman (1977, 1978, 1990) noted in his critique of what we call SITIA conditioning models. For example, Rachman noted that these conditioning models predicted that many people who were subjected to repeated air raids during World War II should have developed intense phobias or other psychiatric casualties. He also noted, however, that air raids in England, Japan, and Germany during World War II did not in fact result in widespread and prolonged panic and terror and quoted Janis (1951, p. 111) as saying "there was a definite decline in overt fear-reactions as the air blitz

continued, even though the raids became heavier and more destructive." Rachman (1990) also cited Janis as having noted, "The bombed population displayed increasing indifference towards the air attacks, and warning signals tended to be disregarded unless attacking planes were in the immediate vicinity" (Rachman, 1990, p. 29). Although this evidence contradicted what was expected by earlier SITIA conditioning models, we do not believe it refutes SIDCA models. For example, Janis's account clearly suggests that the bombed population displayed some degree of fear when warning signals were accompanied by the sight or sounds of attacking planes. In other words, there was some conditioned fear, but it was relatively circumscribed because there were clear danger signals (sight or sounds of attacking planes) in the presence of occasion-setting stimuli (warning signals). Alternatively, it may well have been the case that early on many people did have fear of the warning signals but that these signals lost their fear-provoking properties because they were not reliable signals of danger. That is, gradually all the conditioning accrued to the sight and sound of the attacking planes themselves because they were the most reliable signals of danger. This would certainly be in accord with the Rescorla-Wagner (1972) model of the dynamics of the conditioning process.

Rachman also noted that these conditioning models (i.e., SITIA) could not explain why the fear reactions did not generalize to the sights, sounds, and smells of the cities that were bombed. As already noted, SIDCA models take into account the contextual conditioning principle that when multiple cues are present on a conditioning trial, the most reliable predictor of the US will block the formation of conditioned associations to the less reliable predictors of the US (Kamin, 1968; Mackintosh, 1983; Rescorla & Wagner, 1972; see also Sutton & Barto, 1981). Certainly the sights, sounds, and smells of a city were less reliable predictors of danger than were the sight and sound of an attacking plane. Why the fear reactions did not generalize to the sights or sounds of friendly or commercial planes can probably be explained within the SIDCA perspective by the fact that these other planes were probably not accompanied by the occasion setter of the warning signal. Finally, SIDCA conditioning models explicitly incorporate the principles of "belongingness" or "preparedness" based on evolutionary history into their models (see below). These principles suggest that it is not too surprising that

air raids did not result in widespread and debilitating fears because "air raids, based as they are on sophisticated modern technology, have no evolutionary history, and for this reason human beings are not predisposed to acquire fears during exposures to bombs released from sky-borne craft" (Rachman, 1978, p. 46).

Next we will examine several phenomena suggesting the importance of what happens following conditioning on the level of fear that is maintained over time—another aspect of the dynamic context considered by SIDCA but not by SITIA models. We will first consider the inflation effect (Rescorla, 1974). Rescorla first conditioned a mild fear to a tone by pairing the tone with weak electric shocks. Later, following conditioning, the subjects were randomly exposed to a more intense traumatic US than was involved in the original conditioning. When subjects were subsequently tested for their fear of the CS that had been originally paired with weak shocks, Rescorla found that the fear conditioned response (CR) had been inflated in the direction that would have been expected if the more intense shock had been involved in the conditioning in the first place. To the extent that parallel effects occur in humans, a person who had a conditioning experience with a mildly traumatic event and acquired a fear of nonphobic intensity might be expected to show an increase in that fear, perhaps to phobic intensity, if a noncontingent, highly traumatic experience occurred at a later point in time. Furthermore, Hendersen (1985) showed that the greater the time interval between the original conditioning experiment and exposure to the higher intensity US, the greater the inflation effect. As Hendersen (1985) noted, it is as if the organism has a memorial representation of the original US that can be altered through later experience with other USs and that the malleability of the fear memory increases with time.

A second variable that affects the persistence of acquired fear is whether the organism has control over the termination of the conditioned stimulus. Starr and Mineka (1977) showed that in the context of an avoidance learning paradigm, fear of the CS declines over the course of avoidance learning (e.g., Kamin, Brimer, & Black, 1963) only if the organism has control over CS and US termination. In their experiment, Yoked animals who were not given the ability to terminate the CS or avoid the US themselves did not show the same decline in fear that the Master animals showed, even though both groups experienced the exact same pattern of nonreinforced CSs as

the Master animals' avoidance responses became well learned. This finding underscores the importance of control in producing this attenuation of fear that is seen as an avoidance response becomes well learned (see also Cook, Mineka, & Trumble, 1987, for related results).

We have summarized a number of experiential variables occurring before, during, and after a stressful experience that affect the level of fear that is experienced, conditioned, or maintained over time. These examples illustrate that the second problem with conditioning models from the SITIA tradition is not a problem for SIDCA models. It is true that the factors involved in the origins and maintenance of fears and phobias are considerably more complex than was generally assumed by behavioral learning theorists in the past. Yet these complexities can be understood from the vantage point of contemporary research on conditioning and learning, which points to the crucial importance of SIDCA models. Thus the major problems with SITIA conditioning models in accounting for individual differences in the acquisition of fear are handled simply in SIDCA models by bringing clinical theories about the origins of phobic fears and anxiety states up-to-date with respect to contemporary knowledge about conditioning.

SELECTIVE ASSOCIATIONS IN THE CONDITIONING OF FEARS AND PHOBIAS

The third problem with conditioning models from the SITIA tradition derives from observations that fears and phobias do not tend to occur to a random arbitrary group of objects associated with trauma (see Table 1, point 3). Seligman (1971) and Öhman and colleagues (e.g., 1986; Öhman, Dimberg, & Öst, 1985) have argued that primates and humans may have a biologically based preparedness to associate rapidly certain objects—such as snakes, spiders, water, and enclosed spaces—with aversive events. They have argued that this is because there may have been a selective advantage in the course of evolution for primates and humans who rapidly acquired fears of certain objects or situations that may frequently have been dangerous or posed a threat to our early ancestors (referred to as fear-relevant stimuli). Öhman and his colleagues in Sweden have done an important series of experiments that are generally consis-

tent with this theory. They have found superior conditioning using slides of snakes and spiders as fear-relevant conditioned stimuli and mild shock as an unconditioned stimulus, compared to what is found using more fear-irrelevant CSs such as slides of flowers and mushrooms or geometric figures. The superior conditioning is usually indexed by enhanced resistance to extinction of conditioned electrodermal responses (e.g., Öhman, 1986; Öhman et al., 1985).

More recent studies from Öhman's laboratory have also shown that following conditioning to fear-relevant CSs, even subliminal presentations of these CSs are sufficient to elicit conditioned responses; by contrast, subliminal presentations of fear-irrelevant CSs do not elicit conditioned responses (e.g., Öhman & Soares, 1993). In another experiment it was shown that subjects who were preselected for being highly fearful of snakes or spiders also showed elevated electrodermal responses to masked (subliminal) presentations of slides of their feared object (Öhman & Soares, 1994). Öhman and Soares have argued that such results may provide a theoretical explanation for the irrationality of phobias. "According to this explanation, phobics are unable to control their fear voluntarily because its origin rests in cognitive structures that are not under the control of conscious intentions. This implies that the autonomic arousal, which provides a central component of the phobia, may be already under way when the stimulus information that defines the phobic object reaches conscious, controlled levels of information processing. . . . It is no wonder, therefore, that phobics remain unable to consciously control their fear and revert to escape and avoidance strategies as keys to coping" (Öhman & Soares, 1993, p. 129).

Although the experiments of Öhman and colleagues provide a convincing case that there is differential conditionability of fear to fear-relevant versus fear-irrelevant stimuli, they cannot rule out ontogenetic factors because all subjects had experiences with snakes and spiders before participating in these experiments. To rule out possible ontogenetically based differences in experience with fear-relevant and fear-irrelevant stimuli it is necessary to test for differences in conditionability to fear-relevant versus fear-irrelevant stimuli that are totally novel to the organism.

Cook and Mineka (1989, 1990, 1991) decided to explore this issue with their observational fear-conditioning paradigm—that is, would monkeys who have never had prior exposure to any of the

objects to be used as conditioned stimuli in the observational fear-conditioning paradigm acquire a fear of fear-irrelevant stimuli such as flowers as easily as they do a fear of fear-relevant stimuli such as snakes? To address this question it was necessary to equate the model's fear performance in the presence of flowers with his or her fear performance in the presence of snakes because the major determinant of the level of snake fear acquired by the observer monkeys is the level of snake fear shown by the fearful models (Cook et al., 1985; Mineka et al., 1984b; Mineka & Cook, 1993). To accomplish this matching of fear levels, Cook and Mineka used edited videotapes in which the model was in reality reacting fearfully to a snake, but—through elaborate splicing techniques—it looked as if the model was reacting to the flowers.

There were two groups in this experiment (Cook & Mineka, 1990, Experiment 2). The SN + FL- group watched two videotapes of model monkeys reacting fearfully to toy snake stimuli and nonfearfully to flower stimuli. The FL + SN- group watched two videotapes of model monkeys reacting fearfully to flower stimuli and nonfearfully to toy snake stimuli. The fear performance of the models that each group saw was identical; the groups differed only in whether the fear appeared to be elicited by a toy snake or by flowers. Monkeys in the SN + FL- group did indeed acquire a significant fear of snakes but not of flowers. By contrast, monkeys in the FL + SN- group did not acquire a fear of flowers. Cook and Mineka (1989, Experiment 2) found comparable results when using as CSs two animals that differed in fear-relevance or preparedness; that is, observer monkeys acquired a significant fear of a toy crocodile but did not acquire a fear of a toy rabbit in a similar study of discriminative observational conditioning. Finally, another experiment demonstrated that when the flower and snake stimuli were used as discriminative stimuli in a complex operant appetitive discrimination paradigm, there were no differences in the monkeys' abilities to learn about these stimuli (Cook & Mineka, 1990, Experiment 3). This makes it unlikely that the flowers were simply less salient stimuli than were the toy snakes in the fear-conditioning experiments (see also Cook & Mineka, 1991).

Thus these monkey experiments on preparedness are consistent with those of Öhman and others who have used human subjects to demonstrate superior conditioning of electrodermal responses to fear-relevant stimuli paired with mild electric shock. Both

monkeys and humans seem selectively to associate certain fear-relevant stimuli with aversive outcomes. Because the monkeys used in these experiments had no prior experience with any of the stimuli used in these experiments, the results of these experiments strongly implicate phylogenetic factors in these selective associations. These findings on selective associations are not surprising, however, because it is well-known among contemporary learning researchers that for a given species certain CS-US combinations are more easily learned about than are other CS-US combinations (e.g., LoLordo, 1979a, b; LoLordo & Droungas, 1989), a point that is incorporated by SIDCA but not SITIA models.

PERSISTENCE OF FEARS AND PHOBIAS

Turning now to the fourth problem for fear-conditioning models from the SITIA tradition, we address the question of why some fears are so persistent and even increase with the passage of time (see Table 1, point 4). One partial answer to this question may derive from research on preparedness and selective associations reviewed above. In their research with human subjects, Öhman and colleagues found that responses conditioned to fear-relevant objects such as snakes and spiders are more resistant to extinction than are responses conditioned to fear-irrelevant objects. Indeed, this seems to be the single most robust finding in the human research on selective associations in fear conditioning (McNally, 1987).

This possibility is also supported by the results of a recent experiment by de Jong, Merckelbach, and Arntz (1995), which used an illusory correlation paradigm adapted from Tomarken, Mineka, and Cook (1989). De Jong and colleagues found that subjects' expectancies that shocks would follow spider slides stayed high over the course of a series of trials in which different categories of slides were randomly followed by different outcomes, in spite of the disconfirming evidence that the subjects were receiving. By contrast, their expectancies that shocks would follow slides of weapons declined over the same series of trials. This provides further support for the idea that expectancies of aversive outcomes (one definition of a fear CR) following phylogenetically fear-relevant stimuli such as spiders are especially resistant to disconfirmation. These findings also ex-

tend the results of Öhman and colleagues because de Jong et al. were not using a traditional extinction paradigm in which the CS is repeatedly presented without the US. In the de Jong et al. experiment USs were still occurring but were randomly paired with the different CSs. Extinction of the expectancy for the shock US occurred for the weapons but not for the spiders.

It was not possible to examine the issue of differential extinction to fear-relevant versus fear-irrelevant stimuli in the monkey research on selective associations because one cannot compare extinction to snakes versus flowers or toy crocodiles versus toy rabbits when no conditioning to the fear-irrelevant stimuli occurred in the first place. Nevertheless, other experiments showed that the snake fear seen in our wild-reared monkey models is extraordinarily resistant to extinction (Mineka et al., 1980; Mineka & Cook, unpublished data; Mineka & Keir, 1983). Thus it may be that strong and intense "prepared" fears, such as seen in these monkey experiments, are more resistant to extinction than are fears to fear-irrelevant objects.

DeSilva, Rachman, and Seligman (1977) addressed this issue retrospectively in a study of phobic patients and found no relation between the degree of preparedness of a phobia and the length of time it took to treat the phobia. A retrospective analysis of this issue, however, does not make a compelling case against this hypothesis because the nature and the type of treatment were not controlled. In addition, there was no attempt to equate the subjects with prepared versus unprepared phobias on temperamental variables or on severity of their phobia. What is needed are prospective studies of phobic patients undergoing the same type of exposure therapy whose phobias differ only in their degree of preparedness (and not in severity of the phobia) to see if the phobias to highly prepared stimuli are slower to extinguish than are the phobias to less prepared stimuli; controlling for temperamental variables would also be important.

Another phenomenon that may well contribute to the persistence of fears and phobias over time is the phenomenon of reinstatement of fear (Rescorla & Heth, 1975). This refers to the finding that following extinction of a conditioned fear, the fear can be reinstated simply through exposure to a noncontingent traumatic stimulus. Thus if a person's fear began to extinguish and he or she was accidentally exposed to a nonrelated traumatic event, this might be sufficient to reinstate the original level of fear.

Eysenck (1968) has also argued that fears may incubate, or increase in intensity, with repeated nonreinforced exposures to a CS that elicits fear because he believes that fear itself is a noxious enough state to serve as a US, and a self-perpetuating reinforcement mechanism may thereby cause the fear to incubate rather than extinguish. Although this theory is intuitively appealing and may account for a few anomalous observations in the literature on fear (e.g., the Napakalov effect—see Eysenck, 1968), there are no good studies to support it (see Bersh, 1980, and Mineka, 1985a, for reviews). Two recent studies by Davey and Matchett (1994) do show that human subjects who are either high in levels of trait anxiety or have undergone induced somatic anxiety and are instructed to rehearse the US following conditioning do show transient increases in the strength of their CR. This suggests that simple mental rehearsal of a US can result in increased CR strength, at least under some conditions.

Overgeneralization of fears is also likely to occur with the passage of time after original conditioning. This common clinical observation (e.g., Beck & Emery, 1985) is easily accounted for by research on conditioning conducted in animals that has amply documented that the generalization gradient around a CS+ is much steeper immediately following conditioning than it is some days or weeks later. As reviewed by Riccio, Richardson, and Ebner (1984) and Riccio, Rabinowitz, and Axelrod (1994), this flattening of the generalization gradient is caused not by a decline in fear to the CS+ but rather by an increase in fear to the generalization test stimuli. It seems that with the passage of time, the organism forgets the specific attributes of the CS that was involved in the original conditioning experience and comes to behave fearfully in a wider range of situations than it would have immediately following conditioning. Hendersen (1985) also reviewed evidence that the specific attributes of the US are forgotten with the passage of time, with the similar result that the organism behaves fearfully in a wider range of situations than it would have immediately following conditioning. Mineka and Tomarken have discussed the possibility of a kind of adaptive conservatism to account for such findings (Mineka, 1992; Mineka & Tomarken, 1989; Tomarken, 1988; see also Hendersen, 1985).

In addition, both Hendersen (1978) and Thomas (1979) demonstrated that conditioned inhibitors of fear lose their fear inhibitory properties with the passage of time. That is, although conditioned

excitors of fear are not forgotten with the passage of time, conditioned inhibitors of fear (or safety signals) are forgotten. This may also indirectly affect the maintenance and overgeneralization of fears with the passage of time. Assuming that similar phenomena occur in human classical conditioning, this would suggest that shortly after the onset of a phobia through traumatic conditioning, the phobic individual might have safety signals or CS-s (inhibitory CSs) that would facilitate discrimination of dangerous versus safe places. With the passage of time, however, the CS-s may lose their fear inhibitory properties, with a possible consequence being a loss of discrimination between safe and dangerous places or events and a concomitant increase in generalization of fears (see Mineka, 1992).

To summarize, in this review of specific phobias, we have suggested that the factors involved in the origins and maintenance of fears and phobias are considerably more complex than has often been assumed by past conditioning models from the SITIA tradition. However, these complexities are expected from the standpoint of conditioning models in the SIDCA tradition that consider a large variety of dynamic contextual variables and how they affect the outcome of conditioning experiences. For example, many phobic fears may be acquired observationally rather than through direct conditioning. Furthermore, for humans most fears do not originate out of a single or even a few trials of direct or observational fear conditioning or avoidance learning, occurring in a vacuum, as was often assumed by conditioning models from the SITIA tradition. Instead, there appear to be a multitude of experiential variables that can occur before, during, or following a direct or an observational conditioning experience that interact and affect the amount of fear that is experienced, conditioned, or maintained over time. Results indicating that the nature of the stimulus used during observational fear conditioning can have powerful effects on whether fear is acquired were also reviewed. Observer monkeys showed rapid conditioning of fear to fear-relevant stimuli such as snakes but not to fear-irrelevant stimuli such as flowers. Furthermore, following conditioning, a host of other factors occurring after acquisition of a fear, such as inflation and forgetting of CS and US specificity, can promote maintenance or even exacerbation of fears or an increase in the places in which fears are exhibited.

Social Phobia

As described in the DSM-III-R (American Psychiatric Association, 1987) and DSM-IV (American Psychiatric Association, 1994), people with social phobia fear one or more social situations in which they believe they might do or say something to embarrass or humiliate themselves or in which they may be evaluated. Although some degree of social anxiety is normal and nearly universal, in social phobia it reaches such proportions that it is markedly distressing and/or interferes with functioning. We begin our discussion of conditioning and ethological models of social phobia in the SIDCA tradition by considering social phobia in an adaptive/functional framework. Öhman and his colleagues (Öhman, 1986; Öhman et al., 1985) proposed that social fears evolved as a by-product of dominance hierarchies. The competitive encounters between members of a social group that help to establish dominance hierarchies often involve ritualized displays of threat on the part of the dominant animal and of fear and submissiveness on the part of the defeated animal (see Öhman et al., 1985, for an excellent summary). Öhman and his colleagues also noted that the fear displayed by the submissive animal more often leads to a short dash to get out of the immediate reach of the attacker than to total escape from the situation. They summarized a good deal of psychophysiological research consistent with the notion that, in comparison to people with specific phobias, the fear response seen in those with social phobias is "much more loosely and conditionally concocted, with a less prominent and reflexive role for active avoidance behavior" (1985, p. 141). Moreover, they also noted that the typical age of onset for social phobias (adolescence and young adulthood) coincides with the time when dominance conflicts become prominent.

PREPAREDNESS AND CONDITIONING MODELS OF SOCIAL PHOBIA

In the first extension of the preparedness theory of phobias to the understanding of social anxiety and phobias, Öhman and Dimberg (1978) reasoned that prepared stimuli for social anxiety should involve social stimuli signaling dominance and intraspecific aggres-

sion if social anxiety evolved as a by-product of dominance hierarchies, as they assumed. Because of the strong facial component, including threat or anger expressions on the part of the dominant animal involved in ritualized displays of dominance and submission, Öhman and Dimberg (1978) hypothesized that angry facial expressions would be prepared or fear-relevant stimuli for social anxiety. Consistent with this hypothesis, they found superior conditioning when slides of angry faces were used as CSs relative to the outcome when slides of happy or neutral faces were used.

A particularly fascinating aspect of this literature is that the studies of preattentive activation of prepared conditioned responses (e.g., Öhman & Soares, 1993; Öhman, Dimberg, and Esteves, 1989) have also been applied to the activation of social anxiety. Öhman (1986) found that, following conditioning, conditioned responses could be elicited to subliminally presented CSs when the CSs were angry faces but not when they were happy or neutral faces. These results may help to account for the irrational quality of social phobia in that the emotional reaction can be activated without conscious awareness of the threat cue (see Mineka & Zinbarg, 1995, for further discussion of these studies and their implications for social phobia).

In summary, the work of Öhman and colleagues on preparedness and social phobias suggests that there is an evolutionarily based predisposition to acquire anxiety to angry, critical, or rejecting faces (see Table 1, point 3). Barlow (1988) further argues that social anxieties will be mild or transient for most individuals, and that full-blown social phobia develops only in individuals who are vulnerable for biological and/or psychological reasons to develop what he calls anxious apprehension about future social situations—as would be expected from the perspective of SIDCA models. The biological sources of vulnerability undoubtedly include the temperamental variables discussed below. Moreover, the psychological vulnerabilities seem to stem from the individual's sense of control (or lack thereof) over the environment, as will also be discussed below, in addition to the experiential variables occurring before, during, and after a conditioning experience (see Table 1, point 2).

Nevertheless, important questions remain about the role of such conditioning in the origins of full-blown social phobia (rather than simply conditioning of electrodermal responses). First, what would determine whether a specific or a social phobia would be con-

ditioned when a traumatic event occured? We hypothesize that social phobia is expected if the US occurred in the presence of salient social stimuli such as eating in public or speaking in front of an audience and specific phobia is expected if the US occurred in the presence of an animal such as a snake or a dog or in the presence of salient inanimate cues such as heights or thunder. Given what we now know about the belongingness principle of associative learning (LoLordo, 1979a, b; LoLordo & Droungas, 1989), we would also expect that USs involving perceived embarrassment or humiliation would be more likely to result in the acquisition of social phobias than USs involving physical danger. The fact that posttraumatic stress disorder, rather than social phobia, is considered to be the most common psychiatric sequel when one perceives one's life to be in danger during a sexual assault, an intraspecific trauma, appears to be consistent with this formulation (Kilpatrick & Resnick, 1993).

All of the previous research on preparedness and phobias has used electric shock as USs. This methodological shortcoming may account, at least in part, for Dimberg's (1986) finding that the CRs acquired to slides of the angry face of one person did not generalize to slides of the angry face of a second person. Such specificity clearly is at odds with the clinical features of social phobia because it is rare for individuals with social phobia to be frightened only of a specific individual. We would predict that generalization to be more likely, however, if an angry voice or some aspect of social humiliation or defeat rather than electric shock were used as the US (see Mineka & Zinbarg, 1995, for a more detailed discussion). Indeed, the animal literature on social defeat, to be discussed in more detail below, is consistent with this prediction. But what evidence do we have that social phobias can indeed occur through direct or observational conditioning of fear in the same way as do many specific phobias? It is to this question that we turn our attention next.

DIRECT TRAUMATIC CONDITIONING, OBSERVATIONAL CONDITIONING, AND SOCIAL PHOBIA

Öst and Hugdahl (1981), in a study cited in the specific phobias section, found that 58% of their sample with social phobia recalled di-

rect traumatic conditioning experiences as having played a role in the onset of their social phobia. More recently, Townsley (1992) reported that 56% of a sample with specific social phobia (fear of one or more specific social situations) recalled direct traumatic conditioning experiences as having been involved in the origins of their phobia, although 20% of a control group had also had similar experiences and did not acquire a phobia (see Table 1, point 2).

Although there are methodological limitations of such studies which rely entirely on retrospective recall, we find the convergence between the results of the two studies to be encouraging. In addition, although the finding that 20% of the normal control group from the Townsley study recalled traumatic conditioning experiences may be problematic for a SITIA conditioning account of the origins of social phobias, it is not problematic from the perspective of SIDCA models. That is, it is well-known among contemporary conditioning researchers that CS-US pairings are neither necessary nor sufficient for the development of conditioned fears; contemporary conditioning research suggests that a host of variables occurring before, during, and after a given conditioning experience affect the amount of fear that is acquired and maintained into the future. Thus we interpret the results reported by Öst and Hugdahl and by Townsley as being consistent with SIDCA models given that a large proportion of people with social phobia report that direct traumatic experiences were involved in the onset of their phobia.

What of the individuals who do not recall direct traumatic experiences before the onset of their social phobia—do they constitute evidence disconfirming a learning theory explanation of the etiology of social phobia? (See Table 1, point 1.) Although the work of Mineka and Cook (Mineka, 1987; Mineka & Cook, 1988; Cook and Mineka, 1991) on the vicarious conditioning of snake fear in rhesus monkeys described earlier has never been extended to the acquisition of social anxiety, ethological studies have produced uncontrolled evidence suggesting that such learning does indeed occur. Certainly, it is clear that dominance hierarchies are passed down from one generation to the next in some species. We are particularly intrigued by de Waal's (1989) observations of the intergenerational transmission of dominance hierarchies in rhesus monkeys that included three instances in which infants were adopted. In all three cases, the infant's rank was based on its adoptive parents rather than its biological parents.

Although this evidence is only indirect (de Waal himself acknowledged that more definitive studies ruling out a genetic contribution are needed), it is consistent with the hypothesis that social behaviors relating to dominance and submissiveness can be acquired through observational learning (see Mineka & Zinbarg, 1995, for further discussion).

There are even fewer data regarding the role of observational conditioning experiences than there are for direct traumatic conditioning experiences in the origins of social phobias in humans. In the only relevant study we are aware of, Öst and Hugdahl (1981) found a smaller percentage of subjects with social phobia who recalled vicarious learning experiences as having played a role in the origin of their social phobia (13%) than recalled direct traumatic conditioning experiences (58%). Independent replications of this finding are required before firm conclusions may be drawn, but these results nevertheless suggest that observational conditioning of social fear and phobia can indeed occur in humans.

TEMPERAMENTAL VARIABLES AND SOCIAL PHOBIA

Temperament is another variable in SIDCA models of social phobia that affects the outcome of a socially traumatic conditioning experience and may help explain why only some individuals who undergo such an experience go on to develop social phobia (see Table 1, point 2). In recent years consensus has grown that behavioral inhibition (e.g., Biederman et al., 1990; Gray, 1982; Kagan, 1994; Kagan, Reznick, & Snidman, 1988) is the temperamental construct that is most relevant for the study of anxiety. The behavioral inhibition system is thought to be responsible for inhibiting behavior in response to novelty and signals for either punishment or frustration. Evidence for the existence of a behavioral inhibition system has been found in several species, including cats (Adamec, 1975), dogs (Scott & Fuller, 1965), humans (Kagan, 1989, 1994), rats (Gray, 1982; Hall, 1941), and rhesus monkeys (Chamove, Eysenck, & Harlow, 1972). Scott and Fuller's (1965) investigations are especially noteworthy in that they permitted the study of the genetic basis of behavioral inhibition and found evidence for a substantial genetic contribution to this trait. Of

particular relevance for understanding the origins of social phobia, many of these studies found that behavioral inhibition is related to conspecific stressors in addition to nonsocial stressors (for a more detailed discussion, see Mineka & Zinbarg, 1995). Moreover, there is some evidence that behavioral inhibition is negatively correlated with copulation frequency (Hall, 1941). This evidence is particularly intriguing because social anxiety in people is also negatively correlated with sexual experience (Leary & Dobbins, 1983) and male sexual dysfunction has been conceptualized as a form of social phobia (Barlow, 1986; Barlow, Chorpita, & Turovsky, this volume; Heimberg & Barlow, 1988). Thus it could be argued that the parallels between behaviorally inhibited animals and people with social phobia may even extend to heterosexual interactions.

This brief review of the behavioral inhibition literature suggests the following two hypotheses: social anxiety should covary significantly with nonsocial forms of anxiety, and behavioral inhibition may be the temperamental vulnerability factor common to most if not all of the anxiety disorders, including social phobia. Zinbarg and Barlow's (1992) study of the structure of anxiety and the anxiety disorders produced evidence consistent with these two hypotheses. These analyses revealed a second-order general factor that was loaded on by each of 23 measures tapping various forms of anxiety, including social anxiety as well as several nonsocial manifestations of anxiety. Moreover, discriminant function analysis revealed that each of the six anxiety disordered groups included in the study (representing each of the DSM-III-R anxiety disorder principal diagnoses except posttraumatic stress disorder) obtained significantly higher scores than a no mental disorder control group on the second-order general factor described above and there were few significant differences among the patient groups on this same factor. Certainly, these results provide unambiguous support for the hypothesis that social anxiety covaries significantly with nonsocial anxiety. In addition, although other alternative explanations are plausible, these results are consistent with the hypothesis that the second-order general factor was tapping a core vulnerability common to all the anxiety disorders, including social phobia.

Finally, extrapolating from animal evidence suggesting an inherited basis for behavioral inhibition (Scott & Fuller, 1965), we would expect to find a genetic contribution to the etiology of social

phobia. More specifically, we would predict that the genetic diathesis for social phobia would be common to most, if not all, of the anxiety disorders. This prediction is consonant with Barlow's (1988) conclusion, based on a review of the available genetic evidence, that "what seems to be inherited is a 'vulnerability' to develop *an* [emphasis added] anxiety disorder, rather than a specific clinical syndrome itself" (p. 176) and with recent findings of Kendler and colleagues (1992a).

UNCONTROLLABILITY AND SOCIAL PHOBIA

Numerous studies have demonstrated that uncontrollable stress, but not controllable stress, reduces aggressiveness and increases submissiveness using several different measures, including shock-elicited aggression and competition for limited food resources (e.g., Maier, Anderson, & Lieberman, 1972; Powell, Francis, Francis, & Schneiderman, 1972; Rapaport & Maier, 1978). Williams and his colleagues (1982; Williams & Lierle, 1986) have found similar results using the colony-intruder test, which they argue provides a more ecologically valid measure of social submissiveness. The colony-intruder test involves placing naive intruder rats in an established colony of rats, where they are typically attacked by the dominant male colony resident (see also Blanchard, Takahashi, & Blanchard, 1977). Given that the colony-intruder test involves interactions between strangers, one could also argue that it provides a more ecologically valid analogue to human social phobia because interacting with strangers is a common anxiety trigger in people with social phobia (Scholing & Emmelkamp, 1990).

Although the above studies all used electric shock as the stressor and it seems evident that neither electric shock nor physical pain is involved in the etiology of social phobia, the literature on the effects of social defeat—a more ecologically valid line of research—provides converging evidence implicating uncontrollability in the onset of social phobia. Repeated social defeat, but not repeated victories, leads to increased submissiveness and lowering of position in a dominance hierarchy (e.g., Ginsburg & Allee, 1942; Kahn, 1951; Scott, 1948; Scott & Marston, 1953; Uhrich, 1938). Interestingly, the defeated animals do not appear to learn to be submissive to specific

animals; rather, they appear to display submissiveness in response to the "general deportment of an aggressive individual" (Ginsburg & Allee, 1942, p. 492). Similarly, Uhrich (1938) concluded that "the subordinates do not recognize the dominant (as an individual) but merely flee from any mouse that happens to attack them or assumes a threatening attitude" (p. 402). In this respect, the social defeat literature would appear to offer a better model of human social phobia than the research performed to date examining conditioning to angry faces using electric shock as the US because in those experiments conditioned responses were elicited only by the specific faces used during conditioning (Dimberg, 1986; Öhman et al., 1985).

Not only does repeated social defeat lead to increased submissiveness, as one might expect, but it also appears to produce many of the effects produced by inescapable shock. First, Williams and Lierle (1988) found that repeated social defeat, but not repeated victories, is associated with a similar escape deficit as was reported in the original demonstrations of the "learned helplessness" effect (e.g., Maier, Seligman, & Solomon, 1969; Overmier & Seligman, 1967; Seligman & Maier, 1967). Second, repeated social defeat, relative to repeated victories, also appears to be associated with potentiated conditioned fear responses similar to those associated with inescapable shock relative to escapable shock. Williams and Scott (1989) found that rats that had been defeated and later tested for fear in the presence of odors from an aggressive colony showed significantly more fear than rats that were either defeated and tested with nonaggressive colony odors, undefeated and tested with aggressive colony odors, or undefeated and tested with nonaggressive colony odors. These results have since been replicated by Williams, Worland, and Smith (1990) and were interpreted as indicating that the odors from the aggressive colonies became conditioned fear stimuli for the defeated animals. Williams, Rogers, and Adler (1990) also showed that the defeated animals' fear responses to aggressive colony odors could be extinguished through prolonged exposure to the odors. (See Mineka & Zinbarg, 1995, for further parallels between the effects of social defeat and exposure to uncontrollable shocks.)

Earlier in our discussion of specific phobias, we cited a study by Mineka et al. (1986) demonstrating that extensive experience with controlling appetitive events can have beneficial consequences, serving at least partially to immunize the organism against the ef-

fects of various stressors. One feature of that study that seems particularly relevant for social phobia was the results of separation tests conducted with monkeys from the replication sample. One set of these tests involved taking the monkeys out of their own group one by one and placing them in with another group for several days (intruder separations). In this situation, the Master subjects coped better when serving as intruders in the Yoked group than vice versa. The Yoked host groups also showed more fear/submissive behavior toward the Master intruders than vice versa. These results suggest that an early history of control over appetitive events can not only immunize the organism against inanimate stressors (such as a toy monster and a large novel playroom), as discussed earlier, but also can immunize against social stressors such as interacting with strangers during a time of stress (separation from one's peer group).

The animal evidence reviewed above strongly suggests that perceptions of uncontrollability play a role in the etiology and maintenance of social anxiety. There is some correlational evidence suggesting an association between perceptions of uncontrollability and social phobia in humans. Cloitre, Heimberg, Liebowitz, and Gitow (1992) found that, in comparison to a group of control subjects, both a group of individuals with social phobia and a group with panic disorder had significantly less belief in their ability to influence events in their lives. More specific analyses revealed that this diminished sense of personal control among the group with social phobia was primarily accounted for by beliefs that control over the significant events in their lives is primarily determined by "powerful others." These results have been replicated by Leung and Heimberg (submitted). Although it is impossible to infer the direction of causality on the basis of this correlational data, they are consistent with the animal data from studies that more directly suggest that uncontrollability plays a causal role in the acquisition of heightened levels of social anxiety.

In summary, simplistic SITIA models suggesting that direct traumatic experiences are the only causal factors in the origins of social fears and phobias are inadequate and outdated. SIDCA learning models of social phobia, consistent with the advances in knowledge about the theoretical and empirical foundations of conditioning that have developed over the past 25 years, on the other hand, are capable of accounting for the complexities and individual differences in-

volved in the etiology of social phobia. SIDCA learning models of social phobias take into account the powerful role of experiential variables occurring before, during, and after both direct and vicarious conditioning experiences in determining the outcome of those direct or vicarious learning experiences. These models also address the role of the preparedness of certain cues for social anxiety and acknowledge the role of temperamental variables in putting certain individuals at higher risk than others. Finally, SIDCA learning models of social phobia also include the role of perceptions of uncontrollability over important life events in influencing the development or maintenance of the symptoms of social phobia. (See Table 1 for a summary.)

Panic Disorder and Agoraphobia

People with panic disorder experience recurrent unexpected panic attacks, usually associated with worry or persistent concern about having another attack—a phenomenon called anticipatory anxiety. Most, but not all, people with panic disorder go on to develop some degree of agoraphobic avoidance; that is, they learn to avoid situations in which, if they had a panic attack, escape might be either difficult or embarrassing. Over the years a variety of conditioning models of panic disorder and agoraphobia have been proposed. One of the most widely cited models is that of Goldstein and Chambless (1978), who implicated conditioning both in the development of agoraphobic avoidance ("these clients begin to avoid situations where they fear panic attacks may occur"; p. 54) and in precipitating panic attacks themselves ("Having suffered one or more panic attacks, these people become hyperalert to their sensations and interpret feelings of mild to moderate anxiety as signs of oncoming panic attacks and react with such anxiety that the dreaded episode is almost invariably induced"; p. 55). The conditioning involved in the initiation of panic attacks themselves is *interoceptive conditioning* as described by Razran (1961) in that the conditioned and unconditioned stimuli are both internal bodily sensations and physiological symptoms of intense arousal ("a client's own physiological signs of arousal become the conditioned stimuli for the powerful conditioned response of a panic attack"; p. 55). More recent variants on

this model include Reiss and McNally's expectancy and anxiety sensitivity model (1985; McNally, 1990, 1994; Reiss, 1987) and van den Hout's (1988) interoceptive fear model. Although these models have some important differences, they share in common the idea that people with panic disorder are characterized by fears of interoceptive bodily sensations.

Conditioning models of panic disorder have been criticized for not accounting for the complexity of the cognitive processes associated with panic disorder (see Table 1, point 5). For example, Clark (1988) criticized interoceptive conditioning models of panic disorder on the basis of the observation that contextual factors may prevent individuals from making catastrophic interpretations of bodily sensations and thereby prevent panic. He argued that conditioning models overpredict panic responses because they would seem to have to predict panic attacks every time the initial symptoms of anxiety (the interoceptive CSs) are experienced.

Although Clark's criticism is applicable to SITIA conditioning models, it is less applicable to SIDCA models. Since the seminal work of Rescorla and Wagner (1972; Wagner & Rescorla, 1972), leading theories of conditioning explicitly incorporate compound and contextual conditioning phenomenon (see also Balsam & Tomie, 1985). The key insight of the Rescorla-Wagner model was that the expectation or emotional significance of a given stimulus configuration is based on the aggregate associative strength of all cues present. For example, the summation test for inhibition is based on the premise that conditioned responding to a conditioned stimulus will be less when that stimulus is presented in compound with a conditioned inhibitor. Thus, when a conditioned excitor of fear (such as bodily sensations of anxiety) is presented in compound with a discrete safety signal or in a context that has acquired safety value, less conditioned fear is observed than would be otherwise and this decrement in fear responding is proportional to the strength of the safety association developed in connection with the safety signal or context. This could easily account for why many panic patients are less likely to panic when in the presence of a "safe" or trusted companion or when in a safe place. Recent empirical support for this observation came from a study by Carter, Hollon, Carson, and Shelton (1995), who found that panic patients showed fewer panic symptoms (less self-rated affective distress, fewer catastrophic cogni-

tions, and less physiological arousal) in response to a CO_2 panic provocation challenge if a safe person they brought with them was present than if they were alone with the experimenter.

As noted earlier, a great deal of recent work has been done on a class of stimuli called occasion setters, which in and of themselves do not evoke conditioned responding but which may influence behavior in the presence of a CS (e.g., Holland, 1983; Rescorla, 1988). That is, the presence or absence of the occasion-setting stimulus can turn on, or turn off, conditioned fear to a CS even though the occasion setter itself does not evoke fear. To use a human analogy, if a panic patient just ran up three flights of stairs and noticed his or her heart racing, the setting (running up stairs) may turn off the conditioned fear to the sensations of heart palpitations.

In addition, van den Hout (1988) notes that a potential problem for conditioning models of panic disorder is that they would seem to predict that panic attacks should extinguish given that the internal bodily sensations that constitute the CSs for panic are encountered or experienced more frequently than are panic attacks (see Table 1, point 4). These seemingly nonreinforced "trials" should result in extinction, he argues. But it is important to remember that panic patients often engage in subtle forms of avoidance behavior (such as holding on to a shopping cart when dizzy), which may effectively protect the CS (dizziness) from extinction (Chorazyna, 1962; Salkovskis, 1988).

Recently some attention has been devoted to developing a primate model of panic disorder through the use of biological challenges, including the injection of sodium lactate, which have been found to provoke panic attacks in humans with panic disorder (e.g., Friedman, Sunderland, & Rosenblum, 1988; Sunderland, Friedman, & Rosenblum, 1989). For example, several studies found that lactate produced "temporally circumscribed episodes of agitation, wariness, and motor responses, normally elicited under stressful or threatening conditions" (Friedman et al., 1988, p. 65). Although the paradigms used in such studies have the potential for studying a conditioning model of panic, as of yet this has not happened. Sunderland et al. (1989) did make incidental observations that over the course of the study, the monkeys began to show signs of behavioral disturbance while being restrained and injected, as if they had developed conditioned distress to these warning cues. Unfortunately,

however, the observations were not made in a systematic enough fashion to assure that this was really the result of classical conditioning. Moreover, no attempt has been made using such paradigms to study the possibility of interoceptive conditioning of these paniclike responses.

The second basic research area that may be of relevance for understanding various features of panic disorder and agoraphobia is the literature on the effects of unpredictable and uncontrollable aversive events. When given a choice, animals and people in general prefer predictable to unpredictable aversive events and generally find predictable aversive events to be less stressful (see Mineka & Hendersen, 1985, for a review). Because panic attacks are highly aversive events for most people with panic disorder, one might expect that people with predicted panic attacks would show lower overall levels of anxiety than people with unpredicted panic attacks. A study by Craske, Glover, and Decola (1995) provided partial support for these predictions. They found that for a subgroup of panic patients who experienced a mixture of predicted and unpredicted attacks, anxiety and worry decreased on the day following a predicted panic attack and increased on the day following an unpredicted attack. They noted that these findings are in keeping with some of the animal literature on experimental neurosis as reviewed by Mineka and Kihlstrom (1978), suggesting that the deleterious effects of lack of predictability may be exacerbated for those who have a history of predictability (see also Rapee, Mattick, & Murell, 1986, for additional evidence regarding the role of predictability in mediating the affective reaction to the physical sensations of panic attacks).

The literature on uncontrollable aversive events also has great potential relevance for understanding various features of panic disorder and agoraphobia. As discussed earlier, it is well-known that controllable aversive events result in much lower levels of conditioned fear than do uncontrollable aversive events. Because panic attacks are highly aversive, one would expect that the degree of perceived uncontrollability that a person has over panic attacks would be positively related to the person's levels of agoraphobic avoidance and anticipatory anxiety. In keeping with this prediction, Telch and colleagues (1989) found that a measure of perceived inability to cope with panic attacks predicted levels of agoraphobic avoidance. A re-

lated hypothesis is that perceived control over the initial symptoms of a panic attack may reduce the likelihood that these symptoms will spiral into a full-blown attack. Sanderson, Rapee, and Barlow (1989) reported a very important experiment demonstrating support for this latter prediction. They had 20 patients with panic disorder undergo a CO_2 panic provocation procedure. Half the patients were told that if a light came on while they were inhaling the CO_2, they could turn a dial and the rate of CO_2 infusion would decrease—this was the perceived control group. The other half of the patients were told the same thing, but for this group the light never came on—the no perceived control group. Although none of the patients exercised control, patients in the perceived control group showed lower levels of physiological arousal and were less likely to panic than were those in the no perceived control group. These two studies by Craske et al. (1995) and Sanderson et al. (1989) point to the fruitfulness of pursuing ideas from the research literature on unpredictable and uncontrollable aversive events in further study of panic disorder and agoraphobia, as would be expected from the perspective of SIDCA models.

Generalized Anxiety Disorder

Generalized anxiety disorder (GAD) is characterized by chronic excessive anxiety and worry about a number of events or activities. This state was traditionally described as free-floating anxiety because it was not anchored to a specific object or situation as are specific or social phobias. DSM-IV criteria specify that the anxiety or worry must occur more days than not for at least six months and that it must be experienced as difficult to control. The subjective experience of excessive anxiety or worry must also be accompanied by at least three of the following six symptoms: restlessness or feelings of being keyed up or on edge; a sense of being easily fatigued; difficulty concentrating or mind going blank; irritability; muscle tension; and sleep disturbance. Because we cannot have access to the content of an animal's thoughts, some might argue that animal models of GAD are not feasible (see Table 1, point 6). Many of the other symptoms of GAD, however, have clearly been seen in animal models, and so we

do not dismiss their utility simply because we cannot gain access to the animal's thoughts.

Many of the best examples of symptoms resembling GAD come from the old experimental neurosis literature from the 1930s and 1940s—long before there were separate anxiety disorders—and so this resemblance went unnoticed until Mineka and Kihlstrom's (1978) review of the experimental neurosis literature. Pavlov, Gantt, Liddell, and Masserman all consistently found patterns of behavior in their animals characterized by extreme agitation, hypersensitivity, rapid respiration and heartbeat, restlessness, piloerection, muscular tension, distractibility, and inability to perform previously learned responses. These symptoms were often apparent outside of the experimental situation in which the "neurotic behavior" was induced—in other words, it was generalized, and it was also often very persistent—sometimes even worsening with the passage of time. Thus they seemed to show many of the symptoms of GAD (see also Mineka, 1985a).

In humans there are undoubtedly genetic and temperamental contributions to GAD (e.g., Barlow, 1988; Kendler et al., 1992b), and we have already reviewed evidence in the social phobia section showing that close analogues of the behavioral inhibition phenomenon exist in animals, with the evidence clearly suggesting that reactivity to nonsocial and social stressors is highly correlated. Thus the temperamental diathesis for a variety of the anxiety disorders is probably relatively nonspecific. But what of experiential contributions to GAD?

As discussed by Mineka (1985a), classical and instrumental conditioning models—mostly from the SITIA tradition—do not fare very well, with the possible exception of interoceptive classical conditioning, which long ago was postulated by Russian investigators to be involved in free-floating anxiety, the earlier term for GAD (Razran, 1961). The idea was that various interoceptive CSs could be paired with unpleasant, anxiety-producing USs and thereby elicit anxiety, even though the person was not aware of the occurrence of the CS. Such interoceptive conditioning is known to be highly stable and resistant to extinction compared to exteroceptive conditioning, and because such CSs (and USs) are part of everyday living, their opportunity for playing a role in GAD is reasonable.

Nevertheless, as would be expected from the perspective of

SIDCA models, there are better animal models for GAD, and they derive once again from the literature on unpredictable and uncontrollable aversive events. For the past 30 years numerous theorists have suggested that uncertainty and helplessness are implicated in generalized anxiety. For example, Mandler (e.g., 1972; Mandler & Watson, 1966) argued that anxiety and arousal occur when a response sequence is interrupted and the person has no perceived control over the interruption. Lazarus (1966; Lazarus & Averill, 1972) argued that anxiety occurs when a person perceives a threat whose source is unknown or ambiguous, leaving no clear response options available. Finally, Seligman (1975) argued that unpredictability over important life events—especially negative ones—led to anxiety.

It may seem surprising to some to refer to helplessness as a model of anxiety when 20 years ago Seligman presented it as a model of depression (1974, 1975). Over the past decade several investigators have proposed that it may be a better model of anxiety than of depression (e.g., Maier, personal communication, 27 May 1995; Mineka, 1985a; Barlow, 1988), although given the high comorbidity between the two, this may not be so surprising. For example, Maier and his colleagues have shown that benzodiazepines—antianxiety drugs—administered before a helplessness induction phase prevent helplessness effects from occurring 24 hours later (Drugan, Ryan, Minor, & Maier, 1984; Sherman, Allers, Petty, & Henn, 1979). These results strongly implicate the induction of anxiety as necessary for learned helplessness effects to be observed. Maier has also shown that following a helplessness induction phase the animals show symptoms of anxiety for several days in totally novel environments. In other words, the anxiety generalizes outside the experimental situation and is not tied to the context in which inescapable shock occurred (Peterson, Maier, & Seligman, 1993).

One prominent theory is that exposure to unpredictable events leads to feelings of chronic fear or anxiety because in the absence of a signal for the aversive event, there is also no safety signal telling the organism when he or she can relax and feel safe (Seligman, 1968; Seligman & Binik, 1977). This certainly seems to parallel the uncertainty theme in the anxiety literature, and the helplessness ideas seem to parallel the response unavailability theme in the anxiety literature. It is also of interest to consider the experimental neurosis lit-

erature referred to above in this context. Mineka and Kihlstrom (1978) reviewed that literature and concluded that the two most prominent themes that ran through the myriad paradigms that all seemed to produce this common set of symptoms—many of which are similar to those seen in GAD—are that important life events become unpredictable or uncontrollable or both. That is, buried within each of the experimental neurosis paradigms is evidence that environmental events of vital importance to the organism (food for a hungry animal or shock for a restrained animal) become unpredictable or uncontrollable or both. Thus, as would be expected from SIDCA models, recent empirical and theoretical work on the effects of uncontrollable and/or unpredictable aversive events has clearly had an important impact on our thinking about GAD.

Posttraumatic Stress Disorder

In DSM-IV, the symptoms of PTSD are arranged into three categories labeled reexperiencing, avoidance, and increased arousal. Reexperiencing symptoms include intrusive recollections of the trauma, nightmares, flashbacks, and emotional distress and/or physiological reactivity at exposure to reminders of the trauma. Foa, Zinbarg, and Olasov-Rothbaum (1992) suggested that the avoidance category can be further subdivided into avoidance of trauma-related situations or thoughts and numbing of emotional responsiveness, including restricted range of affect, detachment from others, and decreased interest in activities. Interestingly, findings that Vietnam veterans with PTSD show reduced pain sensitivity when exposed to reminders of combat suggests that numbing may have a literal manifestation as analgesia (Pitman, van der Kolk, Orr, & Greenberg, 1990; van der Kolk, Pitman, & Orr, 1989). Arousal symptoms include disturbed functioning (difficulty sleeping or concentrating), generalized anxiety (exaggerated startle, hypervigilance), and irritability.

It should be readily apparent, even from the brief description above, that the symptom profile for PTSD is the most complex of all the anxiety disorders. Although there is a great deal of symptom overlap between PTSD and the other anxiety disorders (e.g., heightened arousal, hypervigilance, avoidance) and between PTSD and depressive disorders (e.g., decreased interest in activities, diffi-

culties sleeping, problems concentrating), other features of PTSD appear to be more distinctive. Certainly the numbing symptoms appear to be distinctive vis-à-vis the other anxiety disorders, and several authors have suggested that a biphasic alternation between episodes of intrusions and numbing may be the hallmark feature of PTSD (e.g., Foa et al., 1992; Horowitz, 1986; van der Kolk, 1987).

Earlier simple conditioning models of PTSD from the SITIA tradition did not fare very well, in part because they failed to model the complexity of the PTSD symptom profile (e.g., Foa et al., 1989). Despite this complexity of the symptom profile, however, several authors have proposed that the animal literature on unpredictable and uncontrollable aversive events may provide mini-models for PTSD from the SIDCA category that can shed light on some of the mechanisms involved in the development of PTSD symptoms (Foa et al., 1992; Kolb, 1987; van der Kolk, 1987; van der Kolk, Greenberg, Boyd, & Krystal, 1985; Başoğlu & Mineka, 1992). These authors suggested numerous parallels between the effects of uncontrollable and unpredictable stress and PTSD. First, Başoğlu and Mineka (1992) argued that the intense physical stressors used in the animal literature (e.g., electric shocks, near drowning from cold water swims, defeats in physical fighting) closely resemble those in at least one form of human traumatization that often leads to PTSD—torture. In addition, we also note that intense physical harm is often present, or at least threatened, in several other forms of human traumatization associated with elevated rates of PTSD, including child abuse, spouse abuse, and both sexual and nonsexual assault. A second similarity is that the persistent course of the disturbances associated with unpredictable and/or uncontrollable aversive events in animals parallels the chronic course of untreated PTSD (e.g., Başoğlu & Mineka, 1992; Foa et al., 1992; Mineka & Kihlstrom, 1978).

Foa et al. (1992) and Başoğlu and Mineka (1992) argued that a third parallel is that there appear to be compelling similarities between the symptoms of PTSD and the behavioral and physiological disturbances observed in animals exposed to unpredictable and/or uncontrollable aversive stimulation. The most obvious of these similarities is the heightened generalized anxiety and arousal characteristic of both PTSD and animals exposed to unpredictable or uncontrollable aversive stimulation. As noted earlier, the arousal symptoms of PTSD as defined in the DSM-IV include indices of both

disturbed physiological functioning (such as difficulty sleeping) and generalized anxiety (such as hypervigilance and exaggerated startle). In addition, psychophysiological studies have repeatedly shown PTSD to be associated with elevated baseline levels of heart rate and blood pressure (e.g., Blanchard, 1990; Blanchard, Kolb, Gerardi, Ryan, & Pallmeyer, 1986; Davidson & Baum, 1986). These symptoms and characteristics of PTSD appear to be analogous to the potentiated levels of contextual anxiety and disturbed physiological functioning associated with uncontrollable and unpredictable shock reviewed elsewhere (see the section on specific phobias earlier in this chapter; for more detailed discussions see Başoğlu & Mineka, 1992; Foa et al., 1992; Mineka & Hendersen, 1985).

There also appears to be a relatively clear analogy between the avoidance symptoms characteristic of PTSD and the patterns of avoidance behavior displayed by animals subjected to uncontrollable and unpredictable shock. This analogy may not be obvious at first glance given that the most widely known sequelae of uncontrollable, unpredictable shock are deficits in escape and *active* avoidance—the classic "learned helplessness" effect (Overmier & Seligman, 1967; Seligman & Maier, 1967; Maier, Albin, & Testa, 1973). An analysis of avoidance learning would be incomplete, however, without considering passive avoidance in addition to active avoidance. In passive avoidance paradigms, the contingencies are arranged such that the organism must withhold a designated response to avoid punishment, in contrast to active avoidance paradigms in which the organism must emit a designated response to avoid punishment. As we have reviewed elsewhere (Foa et al., 1992; Rush, Mineka, & Suomi, 1982; Zinbarg, Barlow, Brown, & Hertz, 1992), a less widely known effect produced by uncontrollable shock is that it appears to be associated with superior learning of passive avoidance. Thus to restate this analogy more clearly and precisely, the enhanced passive avoidance behavior displayed by animals exposed to uncontrollable shock appears to resemble the avoidance symptoms of PTSD sufferers in which they *passively* avoid entering situations where they might encounter reminders of their trauma.

It is also interesting that child abuse survivors are not only at increased risk for the development of PTSD following adult victimization but are also at increased risk for being victimized as adults (e.g., Feuer & Zinbarg, 1995; Bolstadt & Zinbarg, 1995). Although the

mechanisms mediating this tendency toward revictimization are unclear, one possibility is that this tendency may reflect deficits at escape and active avoidance. That is, when a risky situation is encountered despite passive attempts to avoid danger, the survivor of child abuse may differ from the person who did not suffer child abuse in terms of the motivational or cognitive sets that might facilitate escaping the situation unharmed. Although this hypothesis is admittedly highly speculative at this time, it will be important to test in the future because it suggests another parallel between the symptoms of PTSD and the disturbances associated with the uncontrollability/unpredictability model.

Given the findings demonstrating stress-induced analgesia in PTSD sufferers noted earlier, there also may be an animal analogue of the numbing symptoms of PTSD. As reviewed elsewhere (Başoğlu & Mineka, 1992; Foa et al., 1992; Maier, 1986, 1989), both escapable and inescapable shock produce stress-induced analgesia. In comparison to the analgesia produced by escapable shock, however, that produced by inescapable shock is opiate-mediated, and although it dissipates fairly rapidly, it is reinstatable at least 24 hours later by exposure to several shocks. In addition, inescapable shock leads to hyperactivity to the analgesic effects of morphine not observed after escapable shock, suggesting that inescapable shock sensitizes the opioid system. That there may also be an opioid-mediated analgesia in PTSD was demonstrated in a Vietnam veteran with PTSD who showed analgesia while watching a video of a combat scene; the analgesia was blocked by naloxone—an opioid antagonist (Pitman et al., 1990). This suggests that the numbing experienced by PTSD sufferers may resemble the analgesia associated with inescapable shock in being mediated by endogenous opioid systems. Torture victims also often report analgesia ("feeling numb all over") (Başoğlu & Mineka, 1992). "For example, one survivor said the difficult part of electrical torture was the beginning; after a while he felt numb all over his body and completely dissociated from the situation" (pp. 207–208)—exactly what would be expected based on the animal literature.

Reexperiencing symptoms would appear to be more problematic to model in animals because several of these symptoms (nightmares, flashbacks, intrusive recollections) are dependent on self-report (see Table 1, point 6). However, only one of the five reexperienc-

ing symptoms listed in the DSM-IV is required for the diagnosis of PTSD, and two of these symptoms are emotional distress at exposure to reminders of the trauma and physiological reactivity at exposure to such reminders. Thus nightmares, flashbacks, and intrusive recollections are not necessary for the diagnosis.

Given the considerations outlined above, Foa et al. (1992) argued that distress at exposure to reminders of trauma is central to the construct of reexperiencing. These authors went on to suggest that emotional distress or physiological reactivity at exposure to reminders of trauma, which are not necessarily dependent on self-report, can be reconceptualized as conditioned fear responses. That is, reminders of trauma can be seen as a CS which, viewed from the perspective of contemporary conditioning models, may be seen as a reminder of the US. This interpretation appears to be particularly consistent with Wagner's (1979, 1981) standard-operating-procedures model of conditioning that suggests that a conditioned fear response is a sign that the CS has associatively primed the representation of the US. In other words, Wagner's model suggests that a conditioned fear response results when the US is being reprocessed or reexperienced. Thus, Foa et al. propose that a conditioned fear response can model some of the reexperiencing symptoms and important aspects of the processes underlying reexperiencing. As discussed earlier in the specific phobias section, numerous studies have shown that a CS for inescapable shock produces greater conditioned fear than does a CS for escapable shock, suggesting that, in comparison to controllable stress, uncontrollable stress is associated with greater reexperiencing symptoms.

Before exploring some of the implications that can be derived from the uncontrollable/unpredictable stress model for the understanding of PTSD, it is important to acknowledge that this model has been considered as relevant to several different anxiety and mood disorders. Indeed, we ourselves have suggested that perceptions of uncontrollability and unpredictability play a role in many of the anxiety disorders discussed in this chapter. This may indicate that the uncontrollability/unpredictability model has been overextended. An alternative perspective emerges, however, when we consider the substantial comorbidity and symptom overlap among the anxiety disorders (Moras, Di Nardo, Brown, & Barlow, 1995; Zinbarg & Barlow, 1992) and between anxiety and depression (Alloy, Kelly,

Mineka, & Clements, 1990; Clark & Watson, 1991a and b). Given this overlap, it may be that the literature on uncontrollability and unpredictability illuminates variables relevant to the core features common to the full spectrum of disorders of negative affect.

Acknowledging that this model may have some relevance for many of the anxiety and depressive disorders does not rule out the possibility that it is more relevant for some disorders than for others or that it is relevant in somewhat different ways for the different disorders. Indeed, it has been suggested that this model is most directly relevant for the study of PTSD (Başoğlu & Mineka, 1992; Foa et al., 1992). The claim of special relevance for PTSD has been based on arguments that the entire syndrome produced by uncontrollable or unpredictable stress, including opioid-mediated analgesia, appears most closely to resemble the PTSD syndrome (Başoğlu & Mineka, 1992; Foa et al., 1992) and the physical stressors most used in the animal literature most closely resemble those involved in the onset of at least some cases of PTSD (Başoğlu & Mineka, 1992).

Having now reviewed evidence regarding the similarity of symptoms of PTSD and those produced by uncontrollable and unpredictable stress, what are the implications of the uncontrollability, unpredictability model for understanding other aspects of PTSD? A number of findings that have emerged from the studies on uncontrollable and unpredictable aversive events allow for the generation of more specific hypotheses regarding human PTSD. First, the experience of trauma survivors can be roughly divided into three broad phases: the pretrauma phase, the traumatization phase, and the posttrauma phase (Başoğlu & Mineka, 1992). Accordingly, the literature on the effects of uncontrollable and unpredictable aversive events can be divided into three broad categories that correspond to each of these three phases (see Table 1, point 2). The analogue of the pretrauma phase in the experimental literature involves studies of the pretreatment variables that either immunize or sensitize the organism to the deleterious effects of subsequent exposure to uncontrollable and unpredictable aversive events. The analogue of the traumatization phase in the experimental literature concerns questions of what are the acute reactions to uncontrollable and unpredictable aversive events. Finally, the analogue of the posttrauma phase in the experimental literature involves the study of both the long-term consequences of uncontrollable and unpredictable stress

and the variables that occur following the experience of uncontrollable and unpredictable stress that influence the intensity and duration of the emotional and behavioral disturbances maintained into the future (Başoğlu & Mineka, 1992).

Starting with the traumatization phase, this model suggests the hypothesis that the degree to which traumas are perceived to be uncontrollable and unpredictable is related to the likelihood that a survivor will develop PTSD symptoms, as well as the entire PTSD syndrome. Alternatively, it may be that the more traumas are perceived to be uncontrollable and unpredictable, the more intense and persistent the PTSD symptoms will be. Ethical considerations obviously prohibit studies attempting to manipulate directly perceptions of uncontrollability and unpredictability of traumatic events in humans; therefore, tests of these hypotheses must rely on correlational designs. Although we are not aware of any prospective studies that have examined the ability of perceptions of uncontrollability and unpredictability to predict the later development or maintenance of PTSD symptoms, there is some cross-sectional evidence that the severity of PTSD symptoms is associated with diminished perceptions of controllability. Kushner, Riggs, Foa, and Miller (1993) found that sexual and nonsexual assault survivors who scored lowest on a scale measuring belief in their ability to influence events in their lives received the highest scores on a clinician-rated composite measure of PTSD symptom severity. Bolstadt and Zinbarg (1995) reported similar results using the Rotter Locus of Control Scale. In this study, survivors of a single sexual assault in adulthood who showed the most diminished perceptions of their ability to influence the events in their lives received the highest scores on a self-report version of the composite measure of PTSD symptom severity used by Kushner et al. (1993).

Given the cross-sectional nature of the studies conducted by Kushner et al. (1993) and Bolstadt and Zinbarg (1995), they are in need of replication using longitudinal designs such as assessing perceptions of uncontrollability over trauma and PTSD symptoms shortly after the experience of a trauma (Time 1) and examining the utility of the perception of uncontrollability measure to predict PTSD symptom severity at a later time after statistically controlling for PTSD symptom severity at Time 1. These cross-sectional results sug-

gest that more rigorous tests of the uncontrollability and unpredictability model of PTSD are warranted.

Regarding the traumatization phase, there is one additional particularly intriguing set of findings that may have implications for our understanding of variables during the traumatization phase that can mitigate against the deleterious effects of trauma. Weiss, Glazer, and Pohorecky (1976) found that the expression of aggression may moderate the stress produced by uncontrollable shock. In this study, rats exposed to uncontrollable shock that were allowed to fight with conspecifics during the shocks showed reduced levels of ulceration relative to yoked rats not given the opportunity to fight or display aggressiveness during the shocks. Furthermore, rats exposed to uncontrollable shock and simply allowed to show aggressive postures toward a conspecific even though they were prevented from actual fighting by the presence of a barrier also showed reduced levels of ulceration. Extrapolating from this finding to stress responses to human torture, Başoğlu and Mineka (1992) hypothesized that the amount of trauma inflicted during torture "per se may be less predictive than is the victim's psychological state of resistance and fighting back versus giving up and conceding defeat" (1992, p. 193). In addition, at least one study found that rape victims who physically fight their attacker adapt better following the rape (Chambless, personal communication, 23 April 1995).

Some of the findings from the social defeat literature, discussed earlier in the social phobia section, appear to be consistent with those reported by Weiss et al. (1976). In particular, several studies showed that the analgesia induced by extensive exposure to attack from conspecifics is more highly correlated with the extent to which the animal assumes the characteristic postures of defeat than with the number of bites actually received (Miczek, Thompson, & Shuster, 1982; Rodgers & Hendrie, 1983). These findings converge with those reported by Weiss et al. in suggesting the hypothesis that the trauma survivor's attitude of resistance versus defeat may be an important determinant of the intensity of posttraumatic stress reactions.

As noted above, the experimental literature not only has implications for understanding the traumatization phase itself, but this literature also suggests pretrauma and posttrauma variables that may influence the development or maintenance of PTSD. There are

studies showing that prior experiences with uncontrollable stressors sensitize the organism toward the deleterious effects of a subsequent experience with uncontrollable trauma (e.g., Drugan, Moye, & Maier, 1982; Moye, Hyson, Grau, & Maier, 1983). These findings led Foa et al. (1992) to predict that repeated childhood abuse would predispose survivors to developing PTSD following later traumatization. Consistent with these hypotheses derived from the animal literature, a history of childhood abuse has been found to be associated with greater PTSD severity in response to sexual and nonsexual assault in adulthood (Dancu, Shoyer, Riggs, & Foa, 1991, cited in Foa et al., 1992). Bolstadt and Zinbarg (1995) extended this finding by showing that abuse is particularly associated with greater severity of PTSD symptoms in response to sexual assault in adulthood when the childhood abuse was experienced repeatedly.

By contrast, there are also findings that suggest that immunization against the effects of uncontrollable stress is possible, at least under some circumstances. Numerous studies have shown that a prior history of control can immunize against the deleterious effects of subsequent uncontrollable stress (e.g., Hannum, Rosellini, & Seligman, 1976; Joffe, Rawson, & Mulick, 1973; Mineka et al., 1986; Moye, Cook, Grau, & Maier, 1981; Moye et al., 1983; Seligman & Maier, 1967; Volpicelli, Ulm, Altenor, & Seligman, 1983; Williams & Maier, 1977; see Başoğlu & Mineka, 1992, for a more detailed review). Indeed, experimental work with animals has shown that immunization procedures not only prevent behavioral indices of learned helplessness but can even prevent physiological changes such as opiate-mediated analgesia that occur following exposure to uncontrollable shocks. In addition, early studies by Seligman (1968) and Weiss (1968) showed that predictable stressors often have significantly less aversive impact than do unpredictable stressors, and more recent studies by Overmier (1985) have shown that exposure to predictable but uncontrollable shocks often does not result in the associative deficit of learned helplessness.

In keeping with such findings, Başoğlu and Mineka (1995) tested the idea that prior psychological preparedness for trauma would relate to less perceived distress during torture. In two previous papers, Başoğlu and colleagues (Başoğlu et al., 1994a; Başoğlu, Paker, Tasdemir, Ozmen, & Sahin, 1994b) reported that only 33% of a sample of 55 carefully studied torture survivors who had been po-

litical activists in Turkey before being imprisoned and tortured had ever qualified for a diagnosis for PTSD (based on the lifetime Structured Clinical Interview for DSM-III-R). At the time of the interview—which was an average of five years following their last experience with torture—only 18% qualified for a diagnosis of PTSD. This low rate of PTSD seemed very surprising at first glance given that these torture survivors had experienced an average of 23 different forms of torture and a mean total number of exposures to various kinds of torture of 291. Nevertheless, this was an unusual sample because they were all political activists and it seemed that their political activism was likely to have led them to be more psychologically prepared and better immunized in the sense of having greater perceived control over their environment than a random criminal who had been imprisoned and tortured. To test this hypothesis, Başoğlu and Mineka (1995) developed a psychological preparedness scale that was a combined index of experience exercising control over important life issues and of predictability of being imprisoned and tortured. Results revealed that psychologically more prepared subjects had experienced a greater variety of forms of torture ($r = .29$, $p < .05$) and had more exposures to torture events ($r = .27$, $p < .05$). Yet there was a trend for the more prepared subjects to have given *lower* global overall distress ratings for their entire torture experience ($r = -.23$, $p < .10$). So these results do provide some support for the proposition that psychological preparedness (again, a combined index of experience exercising control over important life issues and of predictability of being imprisoned and tortured) does relate to lower global distress rating. It will still be important, however, to compare these results with those from a sample of prisoners who are not political activists but who have had comparable experiences with torture. Rates of PTSD should be much higher in such a sample, according to the present theory.

Nevertheless, the literature regarding prior experiences with control appears to be complex because other studies have found that having prior experience with control can make the subsequent reaction to uncontrollable stress more severe when control is taken away, relative to what is seen in animals with no prior experience with control (e.g., Hanson, Larson, & Snowdon, 1976; Staub, Tursky, & Schwartz, 1971; Weiss, 1971). These sensitization effects may be related to the superconditioning effect in which greater condi-

tioned fear accrues to a CS if it is presented in compound with a conditioned inhibitor of fear during acquisition training (Rescorla, 1971). This finding suggests that violation of previously held safety associations, such as previously held perceptions of control over one's environment, the beneficial effects of which may be mediated, at least in part, by the safety signal properties associated with the controlling response (Cook et al. 1987; Mineka et al., 1984a), can have particularly deleterious effects on the organism.

There has not yet been a completely satisfactory resolution of the apparent inconsistencies in when prior experience with control has beneficial as opposed to deleterious consequences. Mineka and Kelly (1989) speculated, however, that contextual factors may be critical in mediating the effects of prior control. On the one hand, these authors proposed that prior control may sensitize the organism to the subsequent effects of uncontrollability experienced in the same context where the organism previously had control (maximizing the sense of loss of control). On the other hand, they proposed that prior control may immunize the organism against the effects of future uncontrollable experiences in contexts dissimilar to the context in which the organism previously had control (see Mineka & Kelly, 1989, for a more detailed discussion). Foa et al. (1992) further speculated that if the resolution suggested by Mineka and Kelly (1989) is valid, the underlying mediating mechanism when sensitization is produced may be that prior safety associations are more likely to be violated because the context in which uncontrollability is experienced is the same as the context in which the organism previously experienced control.

The reinstatement effect discussed earlier in this chapter is one example of a posttrauma experience that may influence the course of PTSD symptoms. An interesting study by Rosellini, Widman, Abrahamsen, and Bassuk (1990) suggested that issues of controllability may interact with potentially reinstating events to influence posttrauma emotional reactions. These investigators found that animals with a prior history of uncontrollable, unpredictable shock appear to be predisposed to show an exaggerated reinstatement of anxiety to a CS when a noncontingent US is presented following extinction training with the CS. Clinical observations suggest that processes similar to reinstatement also occur in people with PTSD. For example, Foa and Dancu (1994) described a case in which a woman

who had achieved considerable symptom reduction in therapy for PTSD related to a sexual assault experienced a relapse in her assault-related symptoms after being involved in a car accident. In this case, it appears that the car accident reinstated her assault-related PTSD symptoms. Extrapolating from the results of Rosellini et al., we would expect that trauma victims who most strongly perceive an initial trauma to be uncontrollable and unpredictable would be at greatest risk for experiencing reinstatement of emotional distress after experiencing another trauma not related to the original trauma.

In addition, recent evidence from the animal laboratory suggests that the reexperiencing symptoms of PTSD might be involved in mediating the often persistent course of this disorder. In particular, Maier (27 May 1995, personal communication) has found that the usual time course of some of the classic learned helplessness effects in rats such as the shuttlebox escape deficit (typically 2–3 days) can be prolonged if the rats are simply exposed at 2–3-day intervals to the context in which they had previously experienced uncontrollable stress. That is, simple reminders of the original trauma through exposure to the context in which it occurred seem sufficient to prolong the time period during which learned helplessness deficits can be observed. Currently, results show the effect can be prolonged to at least 12 days. By analogy, such findings suggest that humans who exhibit many reexperiencing symptoms (e.g., flashbacks, nightmares, or simple reexposure to cues for trauma) may be expected to show a more persistent time course for the disorder relative to those with fewer reexperiencing symptoms.

In summary, there appear to be some compelling similarities between the often persistent behavioral disturbances produced by uncontrollable and unpredictable shock in animals and the chronic symptoms of PTSD in humans. In addition, intense physical stressors and stressors such as social defeat used in the animal literature resemble those in at least some forms of human traumatization that often lead to PTSD, including torture, child abuse, spouse abuse, and both sexual and nonsexual assault. These similarities suggest that perceptions of uncontrollability and unpredictability may influence the development and maintenance of PTSD symptoms. Initial studies at the human level have produced evidence that is largely consistent with this hypothesis (Bolstadt & Zinbarg, 1995; Kushner et al., 1993). The animal literature on predictability and im-

munization may also help explain why some people seem to be at reduced risk for PTSD. For example, it may help explain why torture survivors who were political activists showed such low rates of PTSD. Conversely, the animal literature on pretreatment variables that sensitize the organism to the deleterious effects of subsequent exposure to uncontrollable and unpredictable aversive events may help to illuminate why some people are at greater risk for PTSD than others. In addition, several studies reviewed by Clark, Watson, and Mineka (1994) show that conduct disorder or antisocial personality disorder may increase risk for PTSD by increasing exposure to traumatic stressors—possibly through a sensitization process (e.g., Helzer, Robins, & McEvoy, 1987; Kulka et al., 1990). This may be because individuals with conduct disorder or antisocial personality disorder often grow up in chaotic environments that expose them to multiple stressors. Finally, studies of the effects of uncontrollability on the later reinstatement of conditioned anxiety may help to explain relapses of PTSD symptoms related to a prior trauma after experiencing another trauma in the future, and studies of how exposure to reminders of trauma may prolong learned helplessness effects may help to explain some aspects of the persistent course of this disorder.

Obsessive-Compulsive Disorder

Obsessive-compulsive disorder (OCD) is defined in DSM-IV by the occurrence of unwanted and intrusive obsessive thoughts, impulses, or distressing images; these are usually accompanied by compulsive behaviors designed to neutralize the obsessive thoughts or images or to prevent some dreaded event or situation. The compulsive behaviors are repetitive behaviors such as hand-washing, ordering, or checking, or mental acts, such as praying or counting, that the person feels driven to perform in response to an obsession. Conditioning and ethological models of obsessive-compulsive disorder are perhaps more limited in scope than those of some of the other anxiety disorders we discuss here. This is in part because it is probably difficult if not impossible to develop an animal model of obsessions because we do not have access to the content of animals' thought processes. Thus it is more difficult to understand the nature of the particular compulsive behavior analogues the animals engage

in because the nature of the particular compulsive behaviors engaged in is so often closely tied to the content of the obsessions (see Table 1, point 6). In spite of this limitation, a good deal has been written about conditioning and ethological models of obsessive-compulsive disorder, and we will briefly review some of the major findings that are thought to be of relevance.

ANIMAL MODELS OF COMPULSIVE BEHAVIORS

At the most basic level of symptom similarity to human compulsive behaviors, at least five different phenomena in the literature have been proposed as animal models. From the animal learning literature, similarities between well-trained avoidance responses and compulsive behaviors have long been noted (e.g., Mineka, 1985a; Solomon, Kamin, & Wynne, 1953). With extensive training, avoidance responses (to avoid electric shock) become stereotyped in nature and highly resistant to extinction. In addition, Maier's (1949) classic studies of frustration and fixation in rats showed that compulsive, ritualistic behavior could be elicited when rats were repeatedly frustrated as they were faced with insoluble discrimination problems (see Mineka, 1985a, for more extensive discussion).

In the animal behavior literature, it has long been observed that animals subjected to a variety of stressful conditions (such as abnormal rearing conditions or confinement such as in a zoo or animal laboratory) often engage in stereotypic behavior, which is generally defined as any repetitive locomotor behavior involving patterned and rhythmic movement. Relatedly, ritualistic behaviors involve idiosyncratic nonlocomotor stereotyped behaviors such as strumming the mesh of a cage or picking at one's teeth for prolonged periods of time. Such stereotyped and ritualistic behaviors are particularly elevated during times of stress such as during repeated social separations in monkeys (Mineka, Suomi, & Delizio, 1981).

Ethologists have also observed that many species of animals engage in displacement activities under situations of high arousal or conflict; in essence, when a motivated behavior is thwarted or blocked, an animal may suddenly engage in some seemingly unrelated behavior, which is usually highly stereotyped in nature and is called a displacement activity. Some have argued that these activities bear some resem-

blance to the compulsive rituals seen in OCD. For example, Holland (1974) observed that two of the most common displacement activities are grooming and nesting and went onto suggest that these may be functionally related to grooming and tidying rituals seen in OCD (see also Pitman, 1989; Stein, Shoulberg, Helton, & Hollander, 1992). That compulsive rituals in OCD tend to be provoked by the anxiety, discomfort, or distress brought on by obsessive thoughts parallels the occurrence of displacement activities during situations of high arousal or conflict. It is also of interest that in Goodall's (1986) extensive observations of the chimpanzees of Gombe, she noted that social grooming occurs frequently (although not exclusively) during periods when the chimpanzees are frightened, tense, or anxious and that it seemed to help them relax during such periods.

Most recently, Rapoport (1989) argued that compulsive rituals may be fixed action patterns that have been released inappropriately, noting that "the behaviors in OCD resemble misplaced grooming and/or protective rituals" (p. 193) (see also Swedo, 1989). This is a more general argument than the displacement activity argument because fixed action patterns can be triggered by appropriate releasing stimuli at nearly any time—not just during periods of conflict or high arousal. The appeal of this more general model may be that it is better able to account for why the occurrence of compulsive rituals is not always associated with emotional states such as anxiety, frustration, or distress (e.g., Rachman & Hodgson, 1980). But it does not help account for why an emotional reaction often seems to be involved in triggering rituals. Nor have researchers yet been able to identify the key releasing stimuli for such fixed action patterns (Swedo, 1989). In addition, compulsive rituals in OCD are not as fixed and invariant across members of a species as are the fixed action patterns traditionally studied by ethologists. Although rituals may become very stereotyped, there seems to be a greater ontogenetic component to their development than is true for fixed action patterns in lower animals, which may not be too surprising given input from our highly developed neocortex.

ANXIETY REDUCTION THEORY

As reviewed elsewhere (e.g., Mineka, 1985a), for years the anxiety-reduction theory was the dominant alternative to the psychoanaly-

tic theories of OCD (e.g., Dollard & Miller, 1950; Metzner, 1963; Nemiah, 1967; Rachman & Hodgson, 1980; Teasdale, 1974). The essential argument, which is based on Mowrer's (1947) two-process theory of avoidance learning, is that obsessive thoughts or images elicit anxiety or discomfort and therefore motivate the performance of what becomes a compulsive ritual; the ritual is reinforced and strengthened by the reduction in anxiety that ensues. At a descriptive level, this theory, which could be said to derive from what we call the SITIA tradition, does seem to describe what goes on in a majority of cases (see Rachman & Hodgson, 1980, for results supporting the theory).

This theory seems, however, more compelling as a theory of the maintenance of OCD than as a theory of etiology. First, there is little if any evidence that a traumatic conditioning history (direct or vicarious) is involved in the origins of OCD in most cases (Mineka, 1985a; Rachman & Hodgson, 1980). Moreover, there is increasing evidence for a biological basis to this disorder (e.g., Baxter, Schwartz, & Guze, 1991; Baxter, Schwartz, Guze, & Szuba, 1992; Insel, 1990, 1992; Liebowitz & Hollander, 1991; Rapoport & Wise, 1988; Swedo, Leonard, & Rapoport, 1992). By contrast, as a model of the maintenance of the disorder it has considerable heuristic value in accounting for many of the features of day-to-day fluctuations in obsessive-compulsive symptoms.

Two findings from the avoidance learning literature have often been cited as relevant to understanding the high frequency of occurrence of compulsive rituals. Avoidance responses occur more frequently with unsignaled (Sidman) than with signaled avoidance paradigms (Mineka, 1985a; Teasdale, 1974). In unsignaled avoidance the contingencies are arranged such that the aversive stimulus will occur at regular intervals (e.g., once every minute) unless the organism emits a designated response during the intervening time interval; there are no external signals that the aversive stimulus is imminent (as in signaled avoidance) other than the passage of time. One can consider compulsive rituals to be avoidance behavior that is under poor or minimal stimulus control because the provoking stimuli are not easily discriminated as reliable danger signals (e.g., how can one tell if a germ is present, or how does one tell the difference between safe and dangerous dirt?). Relatedly, it is well-known that avoidance learning and efficient avoidance maintenance are facili-

tated by the presence of safety signals or feedback stimuli when the avoidance response is made (e.g., Bolles, 1970; Denny, 1971; Mineka, 1979; Mowrer, 1960; Weisman & Litner, 1969, 1972). These feedback stimuli are thought to acquire fear inhibitory properties and to become sources of positive reinforcement for the avoidance response. Several theorists have argued that such findings are of relevance to understanding the high rate of occurrence of compulsive behaviors because many compulsive rituals can be characterized as having poor feedback or safety-signal properties (e.g., Mineka, 1985a; Rachman & Hodgson, 1980; Teasdale, 1974). In particular, if, as noted above, the particular stimuli provoking the response are poorly defined, it follows that accurate and convincing feedback about their removal will also be difficult to obtain (e.g., if you cannot detect whether germs are present, it is also difficult to determine whether they are gone). Moreover, the dangers that often precipitate checking rituals are often invisible, or intangible, future events, and so it is inherently difficult to know whether one has been successful in avoiding them. As Rachman and Hodgson noted, the obsessive-compulsive "has no assurance that the task is finished (i.e., that the perceived danger has been removed). At best the risks are diminished" (1980, p. 134).

Two more phenomena that have been studied in the avoidance learning literature may also help explain why the frequency of compulsive rituals sometimes shows temporary increases. First, it is well known that CS+s for fear when presented in an unsignaled avoidance situation will cause an increase in the rate of avoidance responding (e.g., Rescorla & LoLordo, 1965). Thus one might expect that rates of ritualizing would increase over baseline levels when novel objects or situations (other than the usual ones) that provoke anxiety or distress are encountered. This is in keeping with clinical observations (Chambless, personal communication, 23 April 1995). Because fear inhibitory phenomena are much more fragile than are fear excitatory effects (Hendersen, 1978, 1985; LoLordo, 1967; Mineka, 1992), however, and because obsessive-compulsives may not have good safety signals for reasons noted above, one may be less likely to see rates of ritualizing decrease from baseline levels, as can be observed when effective CS-s are presented during unsignaled avoidance responding (e.g., Rescorla & LoLordo, 1965). In other words, there may be a much wider range of frightening or distress-

ing situations that could increase rates of ritualizing compared to a relative paucity of inhibitory stimuli or situations that might reduce their rates of occurrence.

The second related phenomenon from the avoidance learning literature of relevance to understanding temporary increases in the frequency of compulsive rituals (Mineka, 1985a; Teasdale, 1974) stems from observations that punishment of avoidance responding very often causes an increase in rate of responding or enhances resistance to extinction of the avoidance response rather than facilitating its extinction (e.g., Brown, 1969; Dean & Pitman, 1991). One popular explanation of this phenomenon starts with the premise that the punishing shocks or free shocks serve to increase the animal's fear or anxiety level. Because the animal has already learned to make the avoidance response in order to reduce the fear of the CS, it continues automatically to make the same response because this response has been strongly reinforced by anxiety reduction in the past. Thus one would expect that presentation of noxious stimulation (USs) could serve to increase the rate of performance of compulsive rituals. The noxious stimuli that could potentially have this effect could range from aversive consequences of excessive performance of the ritual itself (e.g., painful, bleeding hands) to criticism and intolerance expressed by family members. Consistent with such predictions, Steketee and Chambless (1995) have found that high levels of expressed emotion in the families of OCD patients undergoing behavioral treatment predicted a poor treatment outcome.

This also raises an important point about the wide variety and generality of the situations in which compulsive behaviors tend to be elicited. Avoidance models of obsessive-compulsive behavior from the SITIA tradition have sometimes been criticized on the grounds that they cannot account for this broad generalization of contexts in which compulsive rituals occur (see Table 1, point 4). Although it is true that most of the literature on avoidance learning has focused on training one response in one context, there are several important findings considered in the context of SIDCA models which effectively counter this criticism. First, it has been shown that independently established CS+s for fear (conditioned in a different context than the avoidance training context) can immediately acquire control over previously established avoidance responses when the CS+ is delivered for the first time in the avoidance context

(e.g., Rescorla & LoLordo, 1965; Solomon & Turner, 1962). Second, and even more important, are several examples in the literature in which highly idiosyncratic responses (such as jumping over barriers or engaging in idiosyncratic running rituals) trained to reduce fear or frustration in one situation are spontaneously exhibited in a totally different situation when a similar emotional state is elicited through different means (e.g., Fonberg, 1956; Ross, 1964; see Amsel, 1971, and Mineka, 1985a, for extended discussions). Taken together, these experiments provide a model for demonstrating that ritualized behaviors can occur across a wide variety of situations given that a similar emotional state is elicited in each when, and if, a particular response has previously been conditioned to occur in the presence of that state. Similarly, the same displacement activities observed by ethologists can occur in a wide variety of situations that create tension or conflict.

PREPAREDNESS AND THE NONRANDOM DISTRIBUTION OF OBSESSIVE THOUGHTS AND COMPULSIVE BEHAVIORS

The concept of preparedness and selective associations has been applied not only to understanding specific and social phobias but also to obsessive-compulsive disorder (e.g., DeSilva, Rachman, & Seligmen, 1977). The basic observation leading to the extension of this hypothesis to OCD was that the themes of obsessive thoughts and images, as well as the nature of the most commonly observed compulsive rituals, seems to be nonrandom (as for the stimuli and situations that provoke specific and social phobias) (see Table 1, point 3). Moreover, this may be because there had been some evolutionarily based predisposition to have such thoughts and to engage in such behaviors. For example, thoughts about dirt and contamination associated with compulsive washing are so common as to make their occurrence seem nonrandom. DeSilva et al.'s (1977) rating system for the preparedness of phobias was extended to obsessive thoughts and compulsive behaviors. As expected, the investigators found that the content of a great majority of obsessions (as well as of phobias) was rated as prepared. Moreover, the preparedness ratings for the compulsive behaviors for the OCD patients were the highest of all.

DeSilva et al. (1977) also tried to determine whether there was any relationship between the preparedness of the content and behavior of the obsessions and compulsions and any other clinical features of the disorder, such as speed of onset or resistance to treatment. As noted in the specific phobia section, there were numerous problems with drawing firm conclusions from such a retrospective study that produced null results. Moreover, there are even problems with the rationale behind the hypotheses of this study. This part of the study was predicated on important features of Seligman's original preparedness theory (1970, 1971), which postulated that prepared associations should not only be selective in nature (i.e., certain CS-US combinations condition especially well together) but also share other common features, such as speed of learning, and resistance to extinction.

In spite of Seligman's claims that his theory will be of little utility unless it can be shown that prepared or selective associations share these other common features (such as high resistance to extinction and ease of acquisition), others have argued that it is not reasonable to expect this kind of covariance (e.g., Cook & Mineka, 1991; Mineka, 1985a; Rozin & Kalat, 1971; Shettleworth, 1972). For example, there is no reason to think that it would always be adaptive to have all associations that are easily acquired always be difficult to extinguish. For example, it may be adaptive to learn easily the location of a new food source but also easily to forget it in an environment where food sources are frequently changing (Rozin & Kalat, 1971). So, as argued elsewhere, the important insight in Seligman's original preparedness theory of phobias probably centers simply on the concept of selective associability (Mineka, 1985a; Cook & Mineka, 1991). That is, primates and humans seem to be biologically prepared to acquire rapidly fears of certain objects or situations that may once have posed a threat to our early ancestors. Extending the argument to OCD, they may also be biologically prepared to obsess or ruminate about certain topics related to situations that may also have posed a threat to our early ancestors. Moreover, they may also be biologically prepared to perform certain responses (and not others) to reduce emotional states of distress.

What evidence is there for this aspect of preparedness theory? There is ample evidence that animals are predisposed to acquire certain escape or avoidance responses to reduce fear or anxiety. For ex-

ample, years ago, Bolles (1970) systematized observations about avoidance learning in animals and developed what he called a species-specific defense response theory of avoidance learning. According to this theory, animals can easily acquire as learned escape or avoidance responses only ones that resemble those that they would exhibit in the wild under conditions of threat—what he called species-specific defense responses. Other, more arbitrary responses are learned with greater difficulty or sometimes not at all.

With regard to the proposition that fear or anxiety is more easily conditioned to certain stimuli that may have posed a threat to our early ancestors, we have already reviewed evidence that this is the case for specific and social phobias. Although there are no exactly parallel experiments providing a model for OCD, the findings of de Silva et al. (1977) regarding the content and behavior of OCD falling predominantly in the "prepared" range, along with similar observations of Rapoport (1989), provide a solid basis for speculating that very similar arguments may apply. Particularly striking in this regard are Goodall's (1986) observations of the chimpanzees of Gombe, who are "quite fastidious, and if their bodies become soiled with dirt (feces, urine, mud, and so forth) they often use leaves to wipe themselves. They also use leaf napkins to dab at bleeding wounds" (p. 545). In addition, she noted, "The Gombe chimpanzees, in fact, seem to have an almost instinctive horror of being soiled with excrement and only very rarely have been seen to touch feces (their own or another's) with their bare hands. If a chimpanzee accidentally becomes smeared with the feces of another, the offending substance is wiped off carefully with leaves. . . . Mothers usually clean themselves at once if they are accidentally dirtied by the excrement of their infants. . . . If a chimpanzee is accidentally sprinkled with urine (by a companion above him, for example), it too may be wiped off with leaves, but the behavior is not so frantic. . . . Chimpanzees often dab at bleeding wounds with leaves, which they then lick; they may repeat the process many times" (pp. 545–547). These careful observations of the Gombe chimpanzees over many years provide ample documentation that humans' obsessions with dirt and contamination did not arise out of a vacuum but rather have deep evolutionary roots.

In summary, as stated at the outset, there are limitations inherent in animal models of OCD because we cannot know the specific

content of animals' thoughts, as would be necessary to determine if they experience anything resembling human obsessive thoughts or images. As reviewed above, however, there are many examples of compulsive behavior in animals that bear significant resemblance to compulsive rituals in humans. Moreover, although the anxiety-reduction model of OCD may have little bearing on understanding the actual etiology of the disorder, there are nonetheless many findings from the avoidance learning literature that may help to explain important features of the course of the disorder, in particular the fluctuations in intensity of symptoms that is commonly seen. Finally, the literature on selective associability for certain stimuli and for certain avoidance responses helps to illuminate why the content of obsessive compulsives' obsessions usually center around only a few common themes and why their compulsive rituals also usually center around behaviors that closely resemble the fixed action patterns and displacement activities that are commonly seen throughout much of the animal kingdom.

Conclusions

In this chapter we have reviewed conditioning and ethological research from animals and humans that we believe is highly relevant to understanding each of the six different anxiety disorders. Many of the criticisms that have been made of conditioning models in the past have been based on long outmoded contiguity views of conditioning that considered only the effects of stress in isolation in producing anxiety and anxiety disorders—what we have called SITIA (Stress-in-Total-Isolation Anxiety) models. By contrast, contemporary conditioning theory is actually quite cognitive and views conditioning as involving the development of expectancies about the nature of the US. Moreover, there are many dynamic contextual variables that affect the outcome of exposure to stressful life events. The role of such variables is explicitly acknowledged by what we have called SIDCA (Stress-in-Dynamic-Context Anxiety) models. These include temperamental variables, the controllability of the US, the relative predictive value of different CSs in a given situation, prior experiences with a conditioned stimulus, subsequent memory modifications that occur with the passage of time, and others. Evo-

lutionary principles of belongingness or selective associability also tell us a good deal about the nonrandom content of people's phobias, obsessive thoughts, and compulsive behaviors. Finally, findings from the vast animal literature on unpredictable and uncontrollable aversive events have considerable relevance to understanding most of the anxiety disorders, with their relevance for PTSD being especially strong. We urge clinical theorists interested in anxiety and the anxiety disorders to abandon their critiques of outmoded SITIA models and to explore the many promising hypotheses for further understanding of the anxiety disorders that stem from the SIDCA models we have presented here and elsewhere (e.g., Cook & Mineka, 1991; Mineka, 1985a; Mineka, 1992; Mineka & Zinbarg, 1991, 1995).

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